South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 24 May 1958 Vol. 32 No. 21 Kaapstad, 24 Mei 1958
Weekly 2s. 6d. Weekliks 2s. 6d.

AN UNUSUAL CAUSE OF HAEMATEMESIS AND MELAENA

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When the surgeon operates on a case of serious gastro-duodenal haemorrhage and fails to demonstrate the source, he is faced with a difficult decision. Most will agree with Maingot[1] that 'blind' gastrectomy is the procedure of choice, on the grounds that the bleeding arises from multiple acute erosions which are neither visible nor palpable. Attention is drawn here, however, to the biliary tract as a possible source of haemorrhage.

CASE REPORT

A Coloured male aged 53 was admitted with a 2-month history of epigastric pain and loss of weight. The pain recurred intermittently and was severe enough to cause fainting. At times it had been accompanied by vomiting and a few days before admission he had suffered a haematemesis consisting of about 5 cupfuls of bright red blood. For 3 weeks he had noticed that his stools had become black in colour and for a similar period he had noticed dypsnoea on exertion and marked weakness.

The relevant findings on examination were clinical anaemia, confirmed by a haemoglobin estimation of 4·5 g.%, a tender, smooth, firm liver palpable 2 finger-breadths below the costal margin, epigastric tenderness, and altered blood in the stool.

Peptic ulceration was considered to be the most likely cause of the gastro-duodenal haemorrhage and it was agreed that surgical treatment should be carried out as soon as the patient's general condition permitted.

First Operation

On laparotomy (upper mid-line incision) the small and large bowel were found to be full of blood; they appeared to be normal. The stomach was normal. There was superficial scarring of the anterior wall of the first part of the duodenum, with 'cayenne pepper' appearance on light friction, indicating a healed duodenal ulcer. The gall-bladder was tense, reddened and bound down by adhesions—thought to be a subacute cholecystitis.

A Polya ante-colic partial gastrectomy was carried out. The duodenum was inspected after transection and fresh blood was seen to emerge from the lumen—although an ulcer crater was not seen, it was thought that the duodenum was almost certainly the source of the bleeding. It was closed with some difficulty partly on account of the adhesions from the cholecystitis and partly on account of the build of the patient, who was very large and fat.

It was decided that cholecystectomy should not be carried out, as the general condition of the patient was only fair. Accordingly, omentum was brought up to surround the gall-bladder and the abdomen closed.

Post-operative Course. For the following 2 weeks the patient did well, and was being considered for discharge when on the 15th post-operative day he developed severe epigastric pain. This was followed by a melaena stool and the haemoglobin dropped to 8 g.%. The blood was replaced, but the following day he had a further severe haematemesis and melaena requiring 4 pints of blood. Bleeding continued intermittently, more being lost per rectum than by mouth. Before a big bleed, he nearly always complained of severe epigastric pain. He was treated by sedation and blood replacement during the bleeds and the Meulengracht regime in the intervals. By the 28th post-operative day large bleeds were still occurring and it was decided to carry out a laparotomy.

Second Operation

The old incision was excised. The anastamosis appeared to be in order and no other abnormality could be discovered save for the distended gall bladder. This was explored, and when the organ was opened a large quantity of bright arterial blood escaped and continued to do so. The tenseness of the viscus was then seen to be due to the fact that it was full of clots, recent and old. The common duct appeared palpably and visibly normal. Cholecystectomy was carried out as rapidly as possible.

The *post-operative course* was uneventful, save for a minor degree of wound sepsis. When seen in the *follow-up* clinic the patient was well and had suffered no recurrence of bleeding.

Pathological Report on Gall-bladder

'The specimen consisted of the opened gall-bladder. It was 12 cm. in length. The wall showed a generalized thickening to about 3-5 mm. and the mucosa was largely destroyed. An irregularly shaped mass of tissue 5 cm. in its greatest length was also present.

'The section from the detached irregular mass consisted of blood clot with some few epithelial cells.

'The portion from the region of the neck showed a spreading poorly differentiated adenocarcinoma, while others from the adjacent body and fundus showed a diffuse infiltration of malignant cells throughout the wall in the form of a "carcinoma simplex".

'In addition there was a marked secondary infection.'

DISCUSSION

Aird[2] states that 'severe haemorrhage may take origin in the biliary tract and even in an apparently normal biliary tract' but does not elaborate further.

Judd,[3] in 1922, described 4 cases of bleeding duodenal ulcer associated with cholecystitis. As in 3 of these gastro-enterostomy was performed as well as cholecystectomy, it is difficult to conclude that the gall-bladder was the source of the bleeding, for a percentage of duodenal ulcers will heal with the short-circuit operation. In a review of all cases of gall-bladder disease at the Mayo Clinic during 1918–1919, Judd mentioned haemorrhage as a symptom in 2·43%. He quotes Crispin, who found that 5% of his series of cases of gall-bladder disease had a history of gastro-duodenal bleeding.

Deaver,[4] in 1914, reported a case of profuse haematemesis in acute cholecystitis where the blood passed down the common duct to regurgitate into the stomach.

It is rare in recent literature to find cholecystitis mentioned as a possible cause of haematemesis or melaena. Reference to tumour as a cause is even rarer, although one author[5] states, 'Haemorrhage from an ulcerating tumour may be a prominent symptom'. Perhaps it would be advisable in cases where a bleeding point is not demonstrated at operation to investigate the biliary tract more thoroughly—aspiration of the gall-bladder or common duct may on occasions be a profitable procedure. Certainly in the case here described, it would have saved the patient a gastrectomy under emergency conditions.

It would seem that in cholecystitis the bleeding would result from intense congestion of the organ, whereas in tumour actual invasion of the vessels would occur. In this case the growth was invading the cystic artery at the neck of the gall-bladder, the blood then passing down the common duct into the bowel.

Unusual features about this neoplasm of the gall-bladder were the sex of the patient, the absence of calculi in the viscus (present in 90% of cases of carcinoma), and the absence of any visible direct or metastatic spread despite a diffusely spreading, poorly differentiated type of growth.

SUMMARY

1. A case of carcinoma of the gall-bladder is described, presenting as haematemesis and melaena. Confusing features were a healed duodenal ulcer and the presence of a supposed subacute cholecystitis.

2. Disease of the biliary tract as a cause of serious bleeding from the gastro-intestinal tract is discussed and the literature briefly reviewed.

3. The suggestion is made that in cases of gastro-intestinal haemorrhage where the bleeding point cannot be demonstrated at operation, before 'blind' gastrectomy is carried out aspiration of the gall-bladder and common duct should be considered.

Thanks are due to Dr. G. J. Joubert, Superintendent of Somerset Hospital, Cape Town, for permission to publish this case; to Dr. A. Landau for referring this case for surgery; to Mr. L. Chanock, under whose care the patient was treated, and to Mr. M. A. Margo for his advice and assistance at the second operation.

REFERENCES

1. Maingot, R. (1955): *Abdominal Operations*, 3rd ed., p. 334. London: H. K. Lewis.
2. Aird, I. (1957): *Companion in Surgical Studies*, 2nd ed., p. 276. Edinburgh: Livingstone.
3. Judd, E. S. (1922): Ann. Surg., 75, 459.
4. Deaver, J. B. (1914): Surg. Gynec. Obstet., 18, 294.
5. Lewis, D. (1955): *Practice of Surgery*, vol. 7, chap. 2, p. 41. Hagerstown, Md. W. F. Prior.

SECOND CONGRESS OF THE UROLOGICAL ASSOCIATION OF SOUTH AFRICA (M.A.S.A.) CAPE TOWN, 17–19 JULY 1958

A number of papers in the programme hold interest for practitioners not engaged in the practice of urology, and it is hoped that as many as are interested will join in the Congress. A cordial invitation is extended to all practitioners, both general and specialist, to attend the meetings and join in the discussions. All the papers, with one or two exceptions, are of 20 minutes' duration and the time allotted to each subject includes the subsequent discussion. All queries regarding the Congress to be addressed to Dr. P. J. M. Retief, 85 St. George's Street, Cape Town (telephone 6-1821 or 2-9243).

PROGRAMME

Thursday 17 *July*, Physiology Lecture Theatre, Medical School, Observatory, Cape.

9—9.30 a.m.	Opening of Congress: Mr. J. A. Currie, President, Urological Association, and Dr. B. Bromilow-Downing, Dean, Faculty of Medicine, U.C.T.
9.30—10.10 a.m.	Pathology of Bladder Tumours: Dr. C. J. Uys, Department of Pathology, U.C.T.
10.10—10.50 a.m.	Diagnosis of Bladder Tumours: Mr. R. D. Barnes (Cape Town).
10.50—11.30 a.m.	Tea interval
11.30 a.m.—12.30 p.m.	Conservative Treatment of Bladder Tumours: Dr. W. Milner, Albany, N.Y., USA.
12.30—2.30 p.m.	Lunch
2.30—3.10 p.m.	Radical Surgical Treatment of Bladder Tumours: Mr. C. D. Kisner (Johannesburg)
3.10—3.30 p.m.	Tea interval
3.30—5 p.m.	Radiotherapy of Bladder Tumours: Dr. M. Cones (Johannesburg), Dr. R. D. Tucker (Cape Town), and Mr. L. le Roux, Physicist, Groote Schuur Hospital, Cape Town

Friday 18 *July*, Physiology Lecture Theatre, Medical School

9—9.30 a.m.	Large abdominal lumps in children: Prof. J. H. Louw (Cape Town)
9.30—10 a.m.	Translumbar Aortography: Mr. L. Blumberg (Cape Town)
10—10.30 a.m.	Unilateral Renal Disease and Hypertension: Mr. J. N. de Klerk (Cape Town)
10.30—11 a.m.	Tea interval
11—11.30 a.m.	Ileocystoplasty: Mr. L. Jordaan (Pretoria)
11.30—12 noon	Urinary Channel Substitution: Mr. M. Modlin (Cape Town).
12—12.30 p.m.	Post-sacral Neurectomy for Hunner's Ulcer: Dr. W. Milner, Albany, USA.
12.30—2.30	Lunch
2.30—3 p.m.	Electrolyte Imbalances: Prof. L. Eales (Cape Town)
3—3.30 p.m.	Non-surgical Haematuria: Dr. C. Merskey (Cape Town)
3.30—4 p.m.	Tea interval
4—4.30 p.m.	Complications after Prostatectomy: Mr. J. D. Joubert (Cape Town)
4.30—5 p.m.	Late complications of Urinary Tuberculosis: Mr. S. Scher (Cape Town)

Saturday 19 *July*, Groote Schuur Hospital, Observatory, Cape

8.30—10 a.m.	Demonstration, Transurethral Resection of Prostate: Dr. W. Milner, Albany, USA.
10—10.30 a.m.	Tea interval
10.30—11.30 a.m.	Presentation of cases and X-rays
11.30 a.m.—12.30 p.m.	Business Meeting

SOCIAL EVENTS

Thursday evening 17 *July*, 6.30 p.m. *For urologists and their wives* Buffet Supper at Mr. S. Scher's residence, 'Hohenek', Serpentine Road, Oranjezicht, Cape Town.

Friday night 18 *July*. *For urologists and guests* (*wives excluded*) Dinner at Klub Here Sewentien, corner Long and Longmarket Streets, Cape Town. Drinks 7 p.m. Dinner 7.45 p.m. Dress formal.

A Women's Committee will arrange entertainment for wives of visiting urologists. Transport and lunches on Thursday and Friday will be arranged for by the host urologists.

South African Medical Journal: Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL

ANTIBIOTIC FOR MALTA FEVER

With the discovery of the effectiveness of chloromycetin against typhoid fever, of streptomycin against plague and of aureomycin against typhus, these exanthemata have to a large extent been robbed of their terrors.

The appearance of a recent paper[1] calling attention to an antibiotic (cathomycin) effective against Malta fever puts a new look onto another of the serious infective fevers.

Malta fever, Mediterranean fever, undulant fever or brucellosis has been a scourge of temperate climates. This group of fevers, though uncommon in South Africa, is here represented by infection with an organism, *Brucella abortus*, whose animal vector is cattle. Goats not being commonly used for milking in South Africa, typical caprine Malta fever seldom occurs. It is to be hoped that cathomycin will be equally effective against these allied strains of Brucella. While the disease does not ordinarily carry a large mortality, the morbidity is high and only supportive measures have been available to keep the mortality figures down. The new antibiotic will have a very welcome effect in cutting short a long debilitating disease and in abolishing the permanently crippling complication of arthritis. Gost reports a very simple treatment as being completely effective; the drug is dispensed in 0·25 gram capsules and 'one of these is given 4 hourly until the day after the crisis, thereafter 6 hourly for 9 days up to a total of 18 g'. He says: 'The constant effect of cathomycin was remarkable. During the first 5 days of treatment no change is apparent; the pyrexia continues as before, and the symptoms remain just the same, but almost always on the fifth day all the symptoms (headache, insomnia, articular pain, anorexia, sweating, etc.) disappear. Splenomegaly also disappears and by the end of the treatment the spleen has returned to its normal size and shape, no matter how large it was. None of the patients relapsed.'

Side-effects are few, about 25% of the patients showing intolerance in the form of a generalized urticarial rash, but this is treated symptomatically and treatment is persisted with, the exanthem disappearing a few days after the drug is stopped. The leucopenia normally present in undulant fever has in no cases been aggravated and 'so far the urticarial eruption has been the only inconvenience caused by cathomycin'. The author concludes that 'if our findings are confirmed, as we feel sure they must be, we may consider the battle against brucellosis to have been won'.

This most heartening report will focus the clinicians'

VAN DIE REDAKSIE

'N ANTIBIOTIESE MIDDEL VIR MALTAKOORS

Toe dit ontdek is hoe doeltreffend chloromycetin teen maagkoors, streptomisien teen die pes en ouromisien teen vlektifus is, het hierdie uitslagsiektes baie van hul vreesaanjaendheid verloor.

Die verskyning van 'n onlangse verhandeling[1] wat die aandag vestig op 'n antibiotiese middel (katomisien) wat effektief is in die behandeling van Maltakoors, het nuwe hoop gebring in die stryd teen 'n ander van die ernstige besmetlike koorssiektes.

Maltakoors, Mediterranean-koors, spirochetekoors of brucellose was nog altyd 'n plaag wat die gematigde klimaatstreke geteister het. Hierdie groep koorssiektes alhoewel hul nie dikwels in Suid-Afrika voorkom nie, word hier veerteenwoordig deur besmetting met 'n organisme, *Brucella abortus*, waarvan beeste die dierlike draer is. Aangesien bokke maar baie min vir melkdoeleindes in Suid-Afrika gebruik word, kom die tipiese Maltakoors baie selde voor. Dit is te hope dat katomisien net so doeltreffend sal wees teen hierdie verwante Brucella-soorte. Hoewel die siekte nie gewoonlik gepaard gaan met 'n hoë sterftesyfer nie, is die siektegevalsyfer baie hoog, en slegs hulpmaatreëls was beskikbaar gewees om die sterftesyfers laag te hou. Die nuwe antibiotiese middel sal 'n baie welkome uitwerking hê omdat dit die duur van 'n lang, verswakkende siekte sal verkort en die blywende verminkende komplikasie van gewrigsontsteking uit die weg sal ruim. Gost maak in sy verslag melding van 'n eenvoudige behandeling wat heeltemal doeltreffend geblyk het; die middel word opgemaak in 0·25 g. kapsules en ‚een van hierdie kapsules word elke 4 uur toegedien tot die dag ná die keerpunt van die siekte, en daarna elke 6 uur, 9 dae lank, totdat 'n totaal van 18 g. bereik is'.

‚Die konstante uitwerking van katomisien was merkwaardig. Gedurende die eerste 5 dae van die behandeling kan geen verandering waargeneem word nie; die koors hou soos tevore aan en die simptome bly onveranderd, maar gewoonlik verdwyn al die simptome (hoofpyn, slapeloosheid, pyn in die gewrigte, aptytverlies, oormatige sweet ens.) op die vyfde dag. Die miltvergroting neem ook af en teen die afloop van die behandeling is die milt weer normaal van grootte en vorm al was dit ook hoe groot. Geeneen van die pasiënte het 'n heraanval ondervind nie.'

Daar is min newe-effekte; omtrent 25% pasiënte toon gevoeligheid in die vorm van 'n algemene urticaria, maar dit word simptomaties behandel en die behandeling as geheel word voortgesit—die uitslag verdwyn weer 'n paar dae nadat die toediening van die middel gestaak word. Daar was geen gevalle waar die leukopenie wat gewoonlik gepaard gaan met brucellose vererger was nie en ‚dusver was die netelroos die enigste hinderlike gevolg van die toediening van katomisien'. Die skrywer som op dat ‚indien ons bevindings bevestig word, en daarvan is ons seker, kan ons die oorlog teen brucellose as gewonne beskou'.

Hierdie uiters aanmoedigende verslag sal die klinici se

attention more and more on earlier diagnosis; with the knowledge that we can hold the disease and prevent the crippling arthritic complications, the pyrexia of unknown origin which lasts more than 7 days will more frequently be given the specific agglutinating test. The public health authorities may likewise be able to strengthen their organization for securing pure milk supplies, for doubtless the effect of the antibiotic on infected cattle will be fully investigated.

To a small group of people the new discovery is of vital importance; undulant fever is well known to be the most infective organism for laboratory workers and to these especially, the new discovery will give a great deal of comfort.

1. Gost, J. T. (1958): Lancet, I, 191.

aandag des te meer spits op vroeë diagnose; met die wete dat ons die siekte kan stuit en die verminkende komplikasie van gewrigsontsteking kan voorkom, sal die koors van onbekende oorsaak wat langer as 7 dae duur meer dikwels onderwerp word aan die spesifieke agglutinasie-toetse. Die volksgesondheid-owerhede mag dus in staat gestel word om hul organisasie vir die bekom van suiwer melkvoorrade te verbeter, want die uitwerking van die antibiotiese middel op besmette beeste sal sonder twyfel deeglik ondersoek word.

Vir 'n klein groepie mense is die nuwe ontdekking lewensbelangrik; dit is goed bekend dat brucellose die mees besmetlike organisme vir laboratoriumwerkers is, en vir hierdie mense in die besonder sal die nuwe ontdekking baie meer gemoedsrus besorg.

1. Gost, J. T. (1958): Lancet, I, 191

HOSPITAL INFECTION

Infection acquired by patients while in hospital has for ages been a major problem in medicine. Before the days of Semmelweis and Lister obstetrical and surgical wards were often little less than death traps as the result of the spread of virulent septic infection. These terrors lost most of their sting as the result of the practical application of bacteriological knowledge, but even to this day cross infection remains a serious threat, especially in children's hospitals and hospitals for infectious diseases, and even in general medical and surgical wards there is commonly evidence of the spread of disease through the use of instruments, dressings, gloves and other equipment that are inadequately sterilized.

For many years most hospitals have been equipped with a central unit for the sterilization of ward and theatre dressing drums, but it has remained the general practice to decentralize much of the hospital sterilization work in different floors, departments and wards, where the processes used commonly provide no assurance of effective sterilization.

An article on the subject is published in this issue of the *Journal* (p. 537) in which Dr. Wm. Laurie describes the sterilizing unit of Edendale Hospital, Pietermaritzburg, a new 750-bed general hospital. Dr. Laurie points out that 'there is no place in hospital practice for boiling as a method of attempted sterilizing'. A temperature of 100°C (and in many parts of South Africa water boils below 100°) will not kill all spores and viruses; to obtain true sterilization the minimum necessary is 121°C moist heat for 30 minutes, and even this may fail to kill all organisms in dressings etc. that are badly packed in the sterilizer. The viruses of serum jaundice and poliomyelitis are amongst those which may survive mere boiling. The problems presented in the sterilization of hospital equipment are difficult and intricate and require apparatus which can hardly be used except in a central installation, and certainly cannot be duplicated in numerous parts of the hospital. Moreover in order that the plant shall work with full efficiency it is essential that it shall be operated under the close control of a competent bacteriologist and regularly checked by laboratory and other tests.

Syringes[1] are an example of widely used medical implements that are not easily sterilized and at the same time maintained in good working condition and protected from contamination after sterilization. Recently the Nuffield Provincial Hospitals Trust conducted an enquiry and published a booklet[2] on a syringe service for hospitals and hospital groups. Steam heat is not recommended for syringes, the method of sterilization that is favoured being a hot-air oven fitted with a fan to circulate the air.

Those who have had to do with the spread of infectious disease in hospitals, especially amongst children, will agree that its prevention is by no means entirely a question of instruments and dressings. Personal contact between patients and between patients and nurses, droplet infection, infected dust and infected food are active spreaders of infection of sepsis or of infectious diseases which present their own problems. If every patient is provided with his own personal utensils, and the use of utensils in common is stopped, much sterilization (or attempted sterilization) may be avoided. A simple example is to provide every patient with his own thermometer.[3] It is a common experience today for patients to enter hospital with conditions (e.g. osteomyelitis) from which organisms (e.g. staphylococci) are recovered which are resistant to the more commonly used antibiotics. This would appear to be an indication of the spread of infection from patients under antibiotic treatment in hospitals or elsewhere.

1. Editorial (1958): Brit. Med. J., I, 93.
2. *The Planning and Organization of Central Syringe Services* (1957): Nuffield Provincial Hospitals Trust, London.
3. Mirvish, I. (1956): S. Afr. Med. J., 30, 413.

Medical, Dental and Pharmacy Act 1928*: Labelling of Potentially Harmful Drugs.* The Minister of Health after consultation with the South African Medical and Dental Council has made the following regulation with effect from 1 July 1958 (Government Notice No. 613 of 2 May 1958):

1. Every package containing a potentially harmful drug when sold or supplied shall be labelled with the letters 'P.H.D.-M.N.M.' in type of a contrasting colour from that of the remainder of the label; provided this regulation shall not apply to medicines dispensed on the prescription of a medical practitioner, dentist or authorized veterinarian.

STERILIZATION IN HOSPITAL PRACTICE*

WM. LAURIE, D.S.O., M.D., T.D.D., *Deputy Provincial Pathologist, Natal*

In recent years there have appeared in the medical press a significant number of reports on cross-infection in hospital patients. Certain of the outbreaks have been so serious as to necessitate the temporary shutting-down of hospitals or parts of hospitals. In some cases the organisms have been spore-bearers (see below), but by and large the commonest organism implicated has been the staphylococcus. An annotation in the *Lancet* (1957) rightly refers to the problem as a hospital plague. The position has become so disturbing that in some hospitals it is almost routine practice to administer 'prophylactic' antibiotics after operation. This in effect is a return to the antiseptic Listerian techniques of almost a hundred years ago.

As Perkins (1956) points out, the factor responsible for the major percentage of failures in sterilization is the present old-fashioned method of hospital sterilization, i.e. a decentralized system of 'sterilization' on several floors and in several departments by many people. It is a case of everybody's business being nobody's business. The term 'sterilized' or 'sterile' means one thing only, namely that all micro-organisms have been killed, whether spore-bearers or not; this cannot be done by the sterilizing equipment usually supplied to wards.

Adequate sterilization of instruments, syringes, dressings, etc. for use in hospital practice can only be achieved by means of a central sterilizing unit designed to serve the whole hospital, both theatre units and wards. It is stated by Perkins (1956) that this idea of central sterilizing units for hospitals derived from a report issued by the American College of Surgeons in 1928, but in point of fact a form of central sterilizing service has been operative in almost all hospitals for many years in that it has always been customary for ward and theatre drums to be sterilized in one unit. The present so-called 'central sterilizing department' is a logical but surprisingly late extension of this principle. We describe below our experience in the application of this extended system to a large non-European hospital in South Africa.

The hospital, Edendale, is a new 750-bed general hospital. It serves the non-European population of Pietermaritzburg and district, all in the Natal Midlands. The work is roughly similar to that of a hospital of similar size in Britain.

STERILIZING AGENTS

Available methods of sterilization include: moist heat, which kills organisms by coagulation of their proteins; dry heat, thought to sterilize by oxidation; and chemical disinfection, which probably kills by direct poisoning.

Moist Heat

With certain exceptions detailed below moist heat is the most satisfactory and widely used method of sterilization. Dubos (1952) claims that syringes, needles and instruments for minor surgery can be sterilized adequately by boiling for 15 minutes in a dilute solution of washing soda. 'Sterilization' by boiling is the method at present in use in hospitals where each ward and other unit is responsible for its own sterilization. There are serious drawbacks to this method: excluding the obvious and common mistakes of failure to adhere to time and to immerse the equipment in the boiling water, it is to be remembered that at heights such as are found in South Africa water does not boil at 100°C; further, temperatures obtained by boiling water do not kill spores in spore-bearing organisms. Viruses may also not be killed by such temperatures; this applies particularly to the virus of homologous serum jaundice and may apply to poliomyelitis virus (Lenard 1951). In fact American workers no longer use the term 'sterilization' in relation to cleansing by boiling; they have introduced the term 'sanitization' in its place.

There is no place in hospital practice for boiling as a method of attempted sterilizing. To obtain true sterilization it is accepted that the minimum necessary is 121°C moist heat for 30 minutes. Even this will not be sufficient to sterilize the contents of drums which have been badly packed. The steam to be effective must circulate freely to and through the packages since it kills by condensing upon the material to be sterilized. The suggested temperature of 121°C can readily be obtained in the modern type of steam sterilizer fitted with a thermostatic valve to prevent a steam-air mixture. With this type of machine the necessary pressure to reach this temperature is about 17 lb. to the square inch above atmospheric pressure. The steam which condenses on the materials must be dried off after sterilization is complete. Drum containers are bad; the steam penetrates slowly; and the drums are generally not dust-proof after being removed from the sterilizer. All drums should be replaced by the package system. In all methods of sterilization materials to be sterilized should be free from grease and dirt. It is to be remembered that no tubed or bottled equipment or material can be sterilized by moist heat because the steam cannot condense upon the contents.

Steam pressure sterilization is suitable for all except sharp instruments, polythene tubes, blankets, catgut, and rubber goods. Utensils and instruments must be sterilized separately from other equipment and materials.

One query often raised is what happens when a surgeon drops an essential instrument. If the instrument is essential then a second such instrument should always be available sterilized in reserve. If this is not possible then the alternative is to fit an emergency sterilizer in the central supply centre. These emergency sterilizers are high-pressure autoclaves operating at 132°C; they are capable of sterilizing equipment in 3 minutes. To save time and instruments certain hospitals are experimenting with the use of such high-pressure autoclaves for all instruments, not merely for emergency work. This system in theory allows of the turn-over of all trays within 10 minutes, but it has certain disadvantages, e.g.: Firstly, there is no safety margin of time, the slightest cutting-down of time rendering the equipment non-sterile; secondly, the autoclaves in most hospitals cannot carry out this type of work; thirdly, such high temperatures can be used only for instruments and even they deteriorate rather more quickly than with lower temperatures. In other words this 'fast' method is really only of value as a sub-section of the work of a highly organized department.

* Summary of address delivered at a meeting of the Natal Inland Branch of the Medical Association of South Africa, October 1957.

Dry Heat

Dry heat sterilizes by oxidation; it is a much less certain method of killing than pressure steam and its use in hospitals should be limited. Spore-bearing organisms are very resistant to dry heat and viruses are relatively resistant. Perkins (1956) states that the National Institutes of Health, USA, lay down that apparatus to be sterilized by dry heat must be held at 170°C for 2 hours. Such high and long-continued temperatures are highly destructive of equipment.

In this department dry sterilization is used only for sharp instruments such as scissors, blades and cutting needles. These are treated at 160°C for 1 hour. This is equivalent roughly to 121°C moist heat for 30 minutes.

Chemical Disinfection

To quote Perkins (1956): 'In many cases it is apparent that chemical disinfection methods are employed for convenience rather than bactericidal efficiency or surgical safety.' With the exception of certain formaldehyde preparations no chemical disinfection fluid kills spores, and their action on *M. tuberculosis* and some viruses is very variable and uncertain. In our work here we have grown staphylococci in 1/20 solutions of one well-known disinfectant. As with dry heat, organisms vary widely in their resistance to chemical disinfectants. One preparation with a satisfactorily wide and effective spectrum of activity is Hibitane (ACI), discussed by Calman and Murray (1956), but here also this substance fails to kill spore-bearing organisms.

In this department chemical disinfectants are used only for the sterilizing and storing of substances which cannot be treated by heat; e.g. polythene tubing, catgut containers, etc. The mixture used for this purpose is that recommended by Perkins (1956), namely sodium tetraborate (50·0 g.), formaldehyde solution (100 ml.) and distilled water (to 1000 ml.). We have found this to be sporicidal in dilutions up to 1/400. Material immersed in this solution is washed thoroughly in sterile distilled water before use.

METHODS

The perfect system of hospital sterilization is one in which sterile sets are immediately available when required for all ward procedures and for all commoner operations. This can only be achieved by a centralized system of sterilization in a department which replaces each used set of equipment by one sterile set within a short period of time. The contents of each individual set should be planned by the clinicians who are to use the sets; in this department we hold the following range of sets: syringes, gloves, various fluids, dressing sets for individual patients (cutting out drum sterilization), anaesthetic sets, packaged feeding bottles and teats, sets for lumbar puncture, paracentesis, catheterization, rectal examination, transfusion, aspiration, etc. The service is now being extended to include theatre needs.

Gloves are a specially difficult problem in sterilization because of the air trapped in the fingers; the air-steam mixture much reduces the effectiveness of the steam. In spite of this, gloves usually receive less treatment than any other equipment; sterility is sacrificed to keeping satisfactory the texture of the rubber. This is especially unfortunate and dangerous in that the absorbable glove powder now in general use—a starch product—contains many spore-bearing organisms which can only be killed by careful prolonged sterilization. Perkins (1956) recommends a very high temperature and pressure for gloves, namely 20 lb. pressure for 15 minutes, but this is highly destructive and in a large series of tests we have found 10 lb. pressure for 1 hour to be satisfactory, provided the glove powder is first sterilized separately under higher pressure.

Syringes form another major problem in hospital sterilization. Even when sterilization is centralized and controlled, replacement costs are very heavy when glass syringes are used in routine work. Here we use nylon syringes for all ward routine; there are minor disadvantages but they are much outweighed by the great saving in breakages and losses in other ways.

Here it should be mentioned that the system whereby trays are set and sterilized centrally when required is not true central sterilization and represents very little advance on the old decentralized method.

Sterility Test

Perkins (1956) rightly points out that regardless of the degree of mechanical perfection of modern sterilizers, sterilization of the load is still dependent upon correct methods of packaging and correct methods of loading the sterilizers. The modern sterilizer not only exhausts itself of air automatically but also maintains a record of sterilizer-chamber temperature through the whole cycle of operation. Such an arrangement is essential in large-scale work; control of sterilizing by reading of pressures is insufficient; e.g., high pressures can develop without high temperatures in the presence of certain defects of the machine. In all machines without temperature-recording apparatus, and in every type as an occasional check, sterility checks must be employed. We use two methods of checking; the first method is the 'time-temperature' system, where the indicators are tubes (supplied by Becton Dickinson of New Jersey) containing a metal alloy which melts only after the desired temperature has been reached and *held* for the required period of time. One such indicator goes through with each cycle of sterilization. The other method of checking techniques is that of 'test strips'. Such strips consist of slips of absorbent paper which has been soaked in a fluid culture of a safe spore-bearing organism and then allowed to dry. With each cycle of sterilization one such strip goes through, and is then cultured to test whether the spores have been killed. With this method the answer is not available until 24 hours after the material has been sterilized.

OUTPUT

We give below our average monthly output of certain of the more important stock items; the list does not include items recently introduced to the range:

Syringes	18,344
Needles	37,000
Sterile sets for ward work	600
Distilled water, ampoules	640
Distilled water, bottles	584
Sterile normal saline, bottles	592
Drums autoclaved (to be replaced by individual sets)	2,864
Gloves (still a limited circulation only)	800 pairs
Feeding bottles	3,440
Teats	3,440
Glove powder, packets	10,000
Miscellaneous	410
	78,714

Probably the most surprising figure is the tremendous increase in the use of syringes, largely the result of the introduction of antibiotics. From the patient's point of view this increase alone justifies the introduction of central sterilization, for wards cannot adequately sterilize the large numbers of syringes now required in hospital treatment.

STAFFING AND COSTS

Central sterilization is not without its disadvantages; all the hospital eggs are in one basket as regards sterilization. The staff of the central department must therefore be trustworthy, well-trained, permanent staff. The department must function about 18 hours daily. In any large hospital this will necessitate 3 key-staff working shifts. Ex-policemen or army staff on pension are the best type; they can learn their duties in a few weeks. The subordinate staff required are about 6 individuals per 250 beds. This junior staff should not be drawn from trainee nurses; the central sterilizing department is a factory, and nursing staff not only learn very little but are changed so frequently as to interfere with the work of the department. The best individual for this subordinate work is the hospital-porter type.

One difficult staff problem is the question of who should be in charge of the sterilizing department. The person chosen must be medically qualified and the choice would seem to lie between the pathologist, who has much to do with sterility testing, etc., and the medical superintendent's department. A third alternative arises from the suggestion by Colebrooke (1955) that each hospital should have an infection-control officer; this officer would be a good choice for the running of the central sterilizing department.

Space is the next difficulty generally; fortunately, contrary to general belief, there is no need for the central sterilizing department to be anywhere near the operating theatre. Space available anywhere in the hospital area will serve the purpose; for instance, in the rebuilding plans for Guy's Hospital (Annotation, Brit. Med. J., 1957) the central sterilizing department for the whole hospital is placed in the semi-basement of the new 11-storey surgical block. The lay-out needs must include provision for a receiving and wash-up room, a distilled water and saline room, a clean packaging room, a sterilizing room, and a storage and issue room. Perkins (1956) gives detailed diagrams of lay-outs on a basis of 4 square feet per hospital bed. This we would regard as the maximum amount required.

Equipment represents an initial heavy expenditure, much of which is later offset by savings, e.g., a longer life of equipment, etc. Mechanization of processes is important, machines being used for needle cleaning, syringe and glove washing, glove powdering, needle sharpening, etc. The equipment should include large modern steam sterilizers of about 25 cubic feet capacity, with one such sterilizer allowed per 250 beds, subject to a minimum of 2. Glove sterilizers should be a separate fitting, together with a high-pressure sterilizer capable of sterilizing instruments within 3 minutes; this last piece of equipment is used for the instrument which has been forgotten or even as a routine method of treating all instruments.

It must be realized that a central sterilizing department is expensive to launch and expensive to run. The advantages must be considerable to outweigh the disadvantages, even when due allowance is made for the immeasurably greater protection afforded to the patient. Perkins (1956) summarizes the advantages of such a system as being efficiency, economy and safety; the first and the last are self-evident; the question of cost requires analysis. The initial cost of equipping the central sterilizing department with increased stocks of instruments, etc., may be discounted in that the equipment is so much better cared for and lasts so much longer that the savings in the recurrent replacement charges discount the initial costs. The only remaining costing problem is one of staff costs. In a hospital of 750 beds the staff required for sterilizing would be 3 supervisors and 18 subordinate staff, a total wages bill of £450 monthly, the subordinate staff being drawn from Bantu staff at £12 each per month; this does not include the salary of the officer in charge.

The only way this amount can be justified is by savings in wages elsewhere in the hospital. In this hospital of 750 beds an analysis of our monthly production shows it to represent a work total of 30 individuals in wards and theatres. Very roughly, for each person employed in the central sterilizing department 2 nurses can be cut from the ward staff. There are also 3 other important savings to be considered: Firstly, by centralization of sterilization there is a large saving in steam and in routine maintenance by the withdrawing of steam and other sterilizers from theatres and wards; secondly, with sterilized complete sets immediately available for all ward procedures there is a substantial saving in the time of the medical staff; lastly, experience has shown that losses from breakages and certain other causes are much reduced when all equipment and instruments are centrally controlled.

ADDENDUM

Although central sterilization provides certain important safeguards against the spread of infection in hospital, there still remain pressing problems in addition to the question of clean instruments. Among other important sources of sepsis we must remember the following:

Air contamination, especially in theatres, is the cause of a significant amount of sepsis, especially staphylococcal. Blowers *et al.* (1955) quote the closing of a thoracic-surgery unit on this account. Generally speaking, the ventilation system of the theatre is at fault in such 'outbreaks'.

Blankets. Frisby (1957) discusses the problem of the infected hospital blanket. He shows that the average blanket in use harbours 48 million organisms and he shows that ordinary methods of laundering affect only a small proportion of this population of pathogens. He suggests that the answer is a blanket made of some substance which can easily be frequently laundered, e.g. Terrylene.

Gauze masks as used in theatres are a poor precaution against the spread of organisms from the nose and throat, even when worn properly and interlaced with a substance like cellophane. At present the Ear, Nose and Throat department of his hospital is experimenting with a new type of mask.

Infant feeding. The only safe method of preparing infant feeds and feeding utensils is by individual packaging of the utensils, with terminal sterilization of the feeds in a central infant-formula department on the same lines as a central sterilizing department. Pending the establishment of such a system in this hospital the infant bottles and teats are sterilized centrally. This has led to a great reduction in the incidence of infantile gastro-enteritis.

CONCLUSION

The above notes show how unsatisfactory is the situation at present in hospital practice and how well justified is the *Lancet's* description (1957) of hospitals as plague houses. The time has come for us to re-appraise our techniques. In the appraisal we should not overlook the present low standards of work in theatres and wards.

SUMMARY

Details are given of the functioning of a central sterilizing department in a large general hospital. It is shown that only by this method can the hospital patient receive the protection to which he is entitled. The extra expenditure necessary to launch and run such a central unit is more than covered by subsequent savings in staff and equipment.

Mention is also made of other factors to be taken into account in any attempts to control sepsis in hospital practice.

For permission to publish this paper I am indebted to Dr. J. Parker, Director of Medical Services, Natal.

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INTUSSUSCEPTION IN THE ADULT

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Intussusception in the adult is, for several reasons, of considerable interest to the clinician. It is rare compared with the condition in infants, most reports placing the incidence in adults at 5% of intussusceptions at all ages; the aetiological factors are varied; and the clinical presentation varies from chronic abdominal pain to acute intestinal obstruction.

In this article 4 cases are presented from the Coronation Hospital, occurring in the past 2½ years. They illustrate many features of the condition.

CASE HISTORIES

Case 1

A Bantu female aged 33 years, admitted in June 1956 under Mr. B. Lewin. She had had severe attacks of colicky abdominal pain for 1 month. She had vomited after meals for 2 weeks and had frequently passed dark blood in her stools. She had been constipated for 3 months, passing small hard stools, and had lost some weight during 4 months. Two days before admission she experienced another attack of severe colic, associated with absolute constipation.

She was obviously acutely obstructed. She was racked by repeated bouts of colicky abdominal pain, the abdomen was distended, there was visible peristalsis, and loud borborygmi were heard during the bouts of pain. No masses were palpable. The rectum was ballooned but there was no blood on the examining finger. An X-ray showed distended loops of small bowel with fluid levels. There was some free fluid in the peritoneal cavity (Fig. 1).

Naso-gastric suction and intravenous fluids failed to bring about much improvement, and a laparotomy was performed by Mr. Lewin. An ileo-ileal intussusception was found, produced by a circular tumour about 2 inches in diameter and situated about 4 feet from the ileo-caecal junction. The intussusception was easily reduced and the tumour-bearing part of the bowel with 4 inches on either side, was resected. An end-to-end anastomosis was performed. A large lymph gland was dissected off the main mesenteric vessels close to their origin. The patient made an uninterrupted recovery from the operation.

The histological report reads: 'Section of portion of "tumour" from the small bowel shows the presence of slightly oedematous fibrous tissue in which there is a dense focal and diffuse inflammatory-cell infiltration, consisting of plasma cells, lymphocytes and eosinophils. The surface area of the tumour consists of acutely inflamed granulation tissue. Section of the mesenteric gland shows the presence of marked follicular hyperplasia and slight sinus catarrh. Scattered in the medullary area are a moderate number of eosinophils and plasma cells, and scanty lymphocytes. No evidence of malignancy has been observed in the sections examined'. (M. G. Wilcocks.)

Case 2

A Bantu male aged 49 years, admitted under Mr. Lewin in November 1956 with a story of colicky abdominal pain radiating from the right iliac fossa to the right hypochondrium for the short period of 1 week. He had experienced some vomiting a few minutes after meals, but there had been no interference with his bowel action and he had never passed blood *per rectum*.

A large mass was felt in the epigastrium extending to the right from below the xiphisternum. The mass was smooth and its considerable mobility in the axis of the transverse colon suggested the possibility of an intussusception.

Blood chemistry and blood counts were normal except for an ESR of 25 mm. Wintrobe. X-rays of the abdomen revealed no evidence of intestinal obstruction and no other abnormality. A barium enema (Figs. 2 and 3) showed the typical appearances of an intussusception, with the apex in the transverse colon.

At this stage the patient refused all forms of treatment for religious reasons, and discharged himself from hospital. At the time of writing, he is still alive and reasonably well but refuses any further examination or treatment.

Case 3

A Bantu male aged 40 years, was admitted in December 1955 under Mr. S. Kay, with a short history of colicky abdominal pain and vomiting, and absolute constipation of 4 days' duration. The symptoms started after a night of alcoholic excess. He also complained of anorexia, loss of weight, night sweats and productive cough for 2 months.

He was cold, clammy, and dehydrated. The abdomen was distended and rigid and a mass was palpable in the hypogastrium. The mass was also palpable rectally and there was dark blood on the examining finger.

An X-ray of the abdomen showed evidence of obstruction at the level of the terminal ileum and free fluid in the peritoneal cavity. The radiographic appearances of tuberculosis at the left lung apex were confirmed by the finding of acid-fast bacilli in his sputum.

At laparotomy on the day of admission (Mr. A. E. Wilkinson) a gangrenous irreducible ileo-ileal intussusception was found. The proximal small bowel was much distended. The intussusception, situated about 2 feet from the ileo-caecal valve, was resected and an end-to-end anastomosis performed. No cause for the intussusception was demonstrated. Post-operatively the case ran a stormy course, with a swinging temperature, and the patient died on the 12th post-operative day. Unfortunately no autopsy examination could be obtained.

Case 4

A Bantu male aged 41 years, admitted in January 1957 under Mr. Lewin, with a 4 weeks' story of colicky abdominal pain. Diarrhoea was precipitated at the onset of his symptoms by taking castor oil, and it continued intermittently for 3 weeks. He never passed blood *per rectum*. There was no vomiting but he complained of anorexia and loss of weight. Some burning and frequency of micturition were present.

He was thin and anaemic, but not dehydrated. There was no abdominal distension. A freely mobile mass, rubbery hard and smooth, was felt in the epigastrium, extending into the left hypochondrium. There was no blood on the examining finger rectally.

An X-ray of the abdomen showed some gaseous distension of the small and large bowels, but the appearances did not suggest a complete obstruction. A barium enema demonstrated the

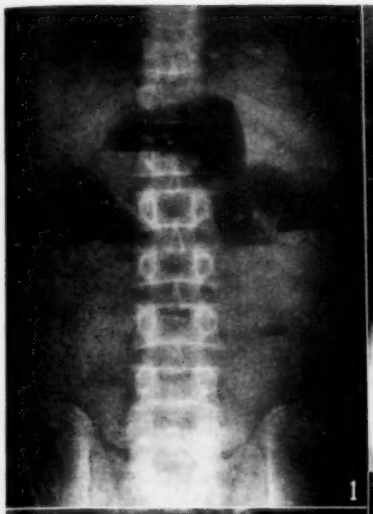

Fig. 1. Case 1. Straight film of abdomen showing the features of small bowel obstruction.

Fig. 2. Case 2. Barium enema showing apex of intussusception in the transverse colon.

Fig. 3. Case 2. Post-evacuation barium enema film showing typical 'coiled spring' effect.

Fig. 4. Case 4. Barium enema showing apex of intussusception near the splenic flexure.

Fig. 5. Case 4. Post-evacuation barium enema film showing the 'coiled spring' seen end-on.

typical appearances of an intussusception with the apex at the splenic flexure (Figs. 4 and 5).

At laparotomy the intussusception was fairly easily reduced, and found to be caeco-colic in type. The apex was formed by an ulcerative lesion with raised edges (palpable through the caecal wall) strongly suggesting a carcinoma. An immediate right hemi-colectomy with end-to-side ileo-colostomy was performed, and the patient made an uneventful recovery.

The histological examination was made by Dr. G. I. Frank: 'Sections from this specimen of caecum and colon show the presence of markedly thickened and very vascular large bowel in which there is much oedema and a diffuse subacute inflammatory reaction. There are areas of acute inflammation, necrosis, and ulceration of both mucosal and serosal surfaces. A few bilharzial ova are present in the submucosa. Sections of lymph glands show the presence of chronic lymphadenitis with reactive hyperplasia and infiltration by plasma cells. There is no evidence of malignancy in the sections examined'.

DISCUSSION

Incidence. Perusal of the literature suggests that intussusception in the adult is rare. Brown and Michels[2] report only 15 cases out of 430,000 new cases at the Cleveland Clinic. In 1925 Lower[6] stated that only 52 cases had been reported in the literature. In the period 1913–1940 only 5 cases of intussusception in the adult were seen at the Peter Bent Brigham Hospital.[1] Donhauser and Kelly[4] reviewed the literature of all cases in the adult reported by American and British writers from 1900 to 1947, and found 665 cases. The appearance of 4 cases in this hospital in the past 2½ years, with at least 1 further case, the records of which could not be traced, suggests that the condition may be commoner than is generally believed.

Age. No age is exempt. In the comprehensive review by Donhauser and Kelly[4] (665 cases), 426 occurred between 15 and 45 years, but there were 65 cases between 61 and 80 years and 2 cases between 81 and 90 years. The 4 cases reported here were aged 33, 40, 41 and 49 years.

Sex. In this small series there were 3 males and 1 female. Donhauser and Kelly found a ratio of 2 males to 1 female in their review of the literature.

Aetiology

It is well known that, unlike the state of affairs in infancy, 'primary' intussusception, where there is no obvious precipitating lesion, is the exception in the adult. In their review of 665 cases, Donhauser and Kelly stated that 158 were said

to be 'primary', but indicated that the figure was probably far too generous. They found the commonest precipitating lesion to be a benign tumour (213 cases), which might be a lipoma, neurofibroma, adenoma, ectopic pancreas, etc. Next in frequency came malignant tumours (123 cases), and then Meckel's diverticulum. The latter occurs particularly in older children and young adults and is characterized by repeated attacks of partial obstruction, frequently by absence of blood in the stools, and frequently by absence of a palpable mass.

In 2 of the cases presented here, the precipitating lesion was of considerable interest. In case 4 an ulcerating bilharzial lesion of the caecum formed the apex of the intussusception. At laparotomy it closely simulated a malignant ulcer and was treated accordingly. In case 1 the macroscopic appearances were those of a benign small-bowel tumour causing an ileo-ileal intussusception. The histological report of a non-specific inflammatory lesion came as a surprise, and the cause of the inflammatory reaction remains obscure. In case 3 the intussusception appeared to be primary, and in the remarkable case 2 the aetiology remains unknown as he steadfastly refused operation. The barium enema did not indicate any causative lesion.

Clinical Presentation

The 4 cases reported here differed in their presentation. The 1st case presented as a subacute intestinal obstruction of 4 weeks' duration which became an acute obstruction 2 days before admission. The 2nd case was never a clinical obstruction at all. The 3rd case presented as a complete acute intestinal obstruction of 4 days' duration, and the 4th case presented throughout with features of subacute intestinal obstruction. This variation in clinical picture, as compared with the fairly stereotyped presentation in the infant, is reflected in the literature. In a recent review, Ferrer[5] found that it was sometimes an acute emergency and sometimes a chronic recurring condition. Donhauser and Kelly[4] state that the symptoms in the adult are so variable that a definite pre-operative diagnosis can only rarely be made. This is probably true for the acute emergencies, but in the more chronic cases the diagnosis should be made in the majority of cases. This is borne out when one considers the cases reported here.

The 4 cardinal signs of intussusception in the infant are intestinal colic, vomiting, the passage of blood *per rectum*, and a mass felt abdominally or rectally, but in the adult it is much less common to find all these features present.

1. *Pain.* All 4 cases in the present series gave a good history of abdominal colic. Donhauser and Kelly[4] found irregular attacks of abdominal colic to be present with few exceptions. Ferrer[5] also found this symptom in 22 of his 25 cases. Other authors, however, have not found it so frequently—Brown and Michels[2] in 9 out of 15 cases, and Deterling *et al.*[3] in 30 out of 40 cases.

2. *Vomiting* was present in 3 of the 4 cases. This agrees with the findings of most authors, but Brown and Michels[2] report it in only 4 of their cases.

3. *Passage of blood per rectum*, as might be expected, was present in the 2 cases presenting acutely, but absent from the 2 more chronic cases. Less than half the cases reported in the literature show this symptom, and Donhauser and Kelly[4] state that, together with tenderness and rigidity, it is infrequent.

4. *Palpable mass.* A mass was palpable in 3 of the 4 cases reported here. Ferrer[5] found a mass in the same proportion of his cases (76%). This is more frequent than the incidence reported by Brown and Michels[2] (one-third to one-half), by Deterling *et al.*[3] (55%), and by Donhauser and Kelly[4] (50%). It is worth noting that in 2 of the cases in which a mass was palpable, its features suggested the possibility of an intussusception.

Nichols[6] emphasizes that, in contrast with other forms of strangulation, intussusception in the adult may occur without tenderness and rigidity. Only 1 of the 4 cases reported here was rigid and distended; in this case a gangrenous intussusception was found at laparotomy.

RADIOGRAPHIC FEATURES

A scout film of the abdomen was normal in only 1 case. In 2 cases there were obvious features of lower small-bowel obstruction, and also evidence of free fluid in the peritoneal cavity. Both these cases were clinically acutely obstructed, and a definite pre-operative diagnosis of the cause of the obstruction was not made. In the last case a straight X-ray showed evidence of partial small-bowel obstruction.

Barium enemata were performed in 2 cases, and the features were pathognomonic, viz:

1. An obstruction to the retrograde flow of barium.
2. A rounded filling defect produced by the apex of the intussusception (Figs. 2 and 4).
3. A 'coiled spring' effect in the post-evacuation films produced by the insinuation of barium between the returning and ensheathing layers of the intussusception (Figs. 3 and 5).

TREATMENT

Reduction by enemata has been enthusiastically advocated in some quarters for intussusception in infants, provided the cases are seen early enough. The method has not found general acceptance, and certainly has little or no place in the treatment of the condition in the adult, the reason being that so few cases are 'primary', and the causative lesion has to be dealt with.

Simple reduction of a primary intussusception in the infant is rarely followed by a recurrence of the condition. In Gross's experience this occurs in about 2% of cases. In the adult we usually have to deal with a precipitating lesion. If there is a causative lesion in the infant, it is probably best left for a subsequent operation.[5] If the condition of the child is very good a large polyp or a Meckel's diverticulum may be excised, but it adds considerably to the risks. In the adult there is more leeway and the cause can usually be dealt with safely. Ferrer,[5] however, feels that if the causative lesion is questionable, it is better to defer the final operation.

If the intussusception is irreducible or gangrenous, several alternative procedures are available. Incision of the neck of the intussusception may make it possible to complete the reduction. In the Jesset operation the ensheathing layer of the intussusception is incised, and the intussusceptum is amputated as high as possible from within the lumen of the intussuscipiens. The intussusception may be fixed *in situ* with interrupted sutures at its neck, a lateral anastomosis performed, and the slough of the intussusceptum allowed to pass spontaneously *per rectum*. None of these methods has found much acceptance among surgeons, and resection in one or two stages, according to the conditions present, is preferable.

In mildly obstructed cases, primary resection-anastomosis is usually feasible. With severer degrees of obstruction a

Mickulicz procedure is indicated. An alternative is Muir's method[7] of decompressing the terminal ileum, after the ileo-transverse anastomosis, by introducing a soft rubber tube or catheter through the closed end of the transverse colon and up the lumen of the terminal ileum for about 6 inches. This tube is brought out through a separate stab incision in the abdominal wall, and is removed in 7–10 days. A further alternative is Woodhall's modification of the Mickulicz procedure: after resection an ileo-transverse anastomosis is performed, but the divided ends of the terminal ileum and the transverse colon are exteriorized. The anastomosis is performed about 2 inches from the divided ends, around which the abdominal wall is closed, leaving 1 inch protruding. These ends are occluded with clamps. If any distension occurs, the clamp on the small bowel is removed. After 7 days an extra-peritoneal closure of the divided ends is performed.

In the ileo-ileal type of intussusception, an enterectomy, including the causative lesion, followed by primary end-to-end anastomosis is indicated if simple excision of the lesion is not feasible. In practice, resection and anastomosis is usually done.

In the 2 ileo-ileal intussusceptions reported here, resection and primary anastomosis was performed, in one because the intussusception was irreducible and gangrenous, and in the other because the causative lesion was thought to be a benign tumour. In the caeco-colic intussusception, the bilharzial lesion of the caecum could not be distinguished from a carcinoma, and a right hemi-colectomy with primary end-to-side ileo-transverse anastomosis was performed.

SUMMARY

1. Four cases of intussusception in the adult are presented. The condition is probably not as rare as is generally believed.

2. The clinical features of the condition are discussed. Presentation differs widely from case to case. There may be merely recurrent attacks of abdominal pain with no clinical intestinal obstruction. There may be recurrent bouts of subacute intestinal obstruction which may terminate in complete obstruction. It may resemble the condition in the infant and present as a sudden acute intestinal obstruction. Vomiting occurs fairly frequently, but passage of blood *per rectum* in less than half of cases. A palpable mass is present in one-half to three-quarters of cases.

3. X-ray of the abdomen will reveal evidence of intestinal obstruction if this is present, while a barium enema shows pathognomonic features if the apex of the intussusception is in the large bowel.

4. In the treatment of the condition, hydrostatic reduction has little or no place in the adult because few cases are primary. At operation the intussusception is reduced if possible, and the causative lesion dealt with according to the conditions present. A simple tumour of the bowel or a Meckel's diverticulum may be excised, but resection and primary anastomosis is usually preferable. In the large bowel, resection in one or two stages, according to the degree of obstruction, is the procedure of choice.

I should like to express my thanks to the following: Mr. B. Lewin for permission to use his cases and for helpful criticism; Mr. S. Kay for permission to use his case; Dr. M. Tonkin, Medical Superintendent of the Coronation Hospital, for permission to publish the cases; Dr. A. D. Bensusan for the photographs; and the S.A.I.M.R. for the histological reports.

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ADDENDUM

Since submitting this paper for publication, the author has encountered a further case in a young Bantu adult male. This patient was admitted under Mr. B. Lewin with generalized abdominal colic following an attack of diarrhoea the day before admission to hospital. There was a doubtful story of passage of blood *per rectum*. He had no further bowel action after admission but he continued to pass a little flatus. Vomiting and abdominal colic persisted. A significant physical sign was shifting point tenderness in the peri-umbilical area. At laparotomy, an ileo-ileal intussusception was found about 4 feet from the ileo-caecal valve. It reduced easily, and the apex was found to be formed by a smooth round tumour projecting into the lumen from the anti-mesenteric border. A wedge resection of the tumour-bearing segment of ileum (about 5 inches in all) was performed, followed by an end-to-end anastomosis. He made a good recovery after the operation. Histological examination showed that the tumour was a polypoid adenoma with mucoid degeneration.

The explanation of the shifting, but extremely localized, tenderness lies in the much greater mobility of the ileo-ileal as compared with the ileo-caecal intussusceptions. No mass could be palpated in this case.

WESSELSBRON VIRUS INFECTIONS IN MAN*

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Wesselsbron virus was first described by Weiss *et al.*[1] as the causative agent of an outbreak of illness in sheep, often associated with abortion, which occurred on a farm in the Wesselsbron area of the Orange Free State during the late summer of 1954–55. The virus was isolated from infected tissues of a lamb that had died 2 days before.

Wesselsbron virus was first isolated from a naturally infected human being, and from mosquitoes (Smithburn *et al.*[2]), about a month after the isolation of the above-mentioned prototype strain. The agent is an arthropod-borne virus (Muspratt *et al.*[3]) which attacks both sheep and man in nature and causes an infection with clinical manifestations.

* The studies and observations on which this paper is based were financed jointly by the South African Institute for Medical Research, the Poliomyelitis Research Foundation, the South African Council for Scientific and Industrial Research, and the Rockefeller Foundation, and were conducted with the collaboration of the Union Health Department.

The aim of this paper is to record 2 further cases of natural infection by Wesselsbron virus in human beings.

Materials and Methods

The procedures and techniques that were employed in the laboratory for the diagnosis of Wesselsbron virus infection in the two human cases were essentially the same as those described by Smithburn *et al.*[2]

Clinical observations were limited to a history and physical examination without clinical pathological investigations.

RESULTS

The two cases of Wesselsbron-virus infection to be described in this report were in personnel who undertook field investigations during an outbreak of illness among sheep in the Middelburg district of the Cape Province in the late summer of 1957. The Director of Veterinary Services had stated that Wesselsbron virus had been isolated from the tissues of a sheep which died in the area and these two individuals, one from the South African Institute for Medical Research and the other from the Medical Ecology Centre of the Union Health Department, undertook to study the arthropod vector or vectors active in the epizootic. An account of the disease outbreak and a description of the topography of the Middelburg region has been published in an article reporting the isolation of an agent other than Wesselsbron virus which was made following these field investigations. This agent has been designated Middelburg virus (Kokernot *et al.*[4]).

After their arrival in the area these two individuals (de M. and P.) proceeded to catch mosquitoes, from which Wesselsbron and Middelburg viruses were subsequently isolated (Kokernot *et al.*[4]). During these operations both men were frequently bitten by mosquitoes. Two days after his arrival de M. performed a post-mortem on a lamb that had died after a short illness and a second autopsy on a ewe that had died in convulsions.

On the morning of the 5th day after his arrival in the Middelburg area de M. awoke feeling stiff and sore, especially at sites of old traumata, which included knee, ankle, left shoulder and back. He had a severe frontal headache with a feeling of pressure behind the eyes. The headache was not relieved by taking 2 aspirin tablets. His temperature at the time was 99°F; there was a further rise to 101°F by mid-afternoon. In the late afternoon, when his temperature was 100·5°F, a blood specimen was taken. During the day he was anorexic and only took fluids. By evening the muscular and joint pains had mostly gone and though he still had a headache it was less intense. The feeling of pressure behind the eyes continued. The next day the patient felt much better and although febrile in the early morning his temperature returned to normal except for a slight rise at midday. He was examined by a doctor that evening, who was unable to elicit any significant physical findings.

The blood specimen was kept on water ice for 3 days before the serum was separated and inoculated intracerebrally into newborn and adult mice for the purpose of attempted virus isolation. Of the 6 adult mice inoculated with the patient's serum, one was dead from a non-specific cause the day after inoculation and another on the 12th day. An attempt at brain passage of this second mouse was unsuccessful. The other 4 mice remained well. Inoculated newborn mice sickened on the 8th and 9th day and brain passages were made from them which led to the establishment of a transmissible agent in both newborn and adult mice. This agent was designated H 112, corresponding to the accession number given to the field specimen.

Sixteen days after initial inoculation of the patient's serum into mice a re-inoculation of the serum (which had been stored in a mechanical deep-freeze) into 6 newborn mice was done. All 6 mice either died or sickened between the 8th and 13th days. Re-isolation of the agent was accomplished.

In an adult-mouse intracerebral neutralization test with adapted H 112 virus against pre- and post-inoculation Wesselsbron-virus sheep sera, the latter neutralized approximately 5·9 logs of the virus. These results indicated that H 112 virus strain is identical with or closely related to Wesselsbron virus.

An intracerebral neutralization test in adult mice with H 112 virus was done against heat-inactivated acute- and convalescent-phase sera obtained from de M. The test showed a sharp rise in antibodies during convalescence of the donor and confirmed the origin of the virus from him. The results of this test are shown in Table I.

TABLE I. RESULTS OF INTRACEREBRAL ADULT-MOUSE NEUTRALIZATION TEST WITH H 112 VIRUS AGAINST THE ACUTE AND CONVALESCENT SERA OBTAINED FROM THE PERSON FROM WHOM THE VIRUS WAS ISOLATED

Serum with date of bleeding*	*Dilution of virus*	*Number of mice*		*Calculated titre of virus 1 to :*
		Died	*Survived*	
Acute-phase 7.4.57	10^{-4}	6	0	500,000
	10^{-5}	6	0	
	10^{-6}	1	5	
	10^{-7}	1	5	
	10^{-8}	0	6	
Convalescent-phase 24.4.57	10^{-2}	1	5	63 or less
	10^{-3}	2	4	
	10^{-4}	0	6	
	10^{-5}	0	6	

* Sera heat-inactivated for 30 minutes at 60°C before testing.

The second individual (P.) participated in the same activities as de M. with the exception that at no stage did he handle living or dead infected animals. He was, however, frequently bitten by mosquitoes while engaged in mosquito catching. The evening of the same day that de M. awoke feeling ill, i.e., the 5th day after their arrival in the Middelburg area, P. felt a sudden headache and pain in his back and left shoulder while working at a catching site. Shortly afterward his temperature was found to be 101·6°F. During the night he suffered rigors and perspired profusely. The following day his febrile reaction continued and he too complained of pain behind the eyes as well as generalized body pains. The patient experienced great discomfort in flexing his limbs owing to muscular pains. He also complained of a pain high up in his abdomen. His temperature at this stage was 98·8° and it was then that a blood specimen was taken for possible virus isolation*. The next day the patient's temperature was normal but he still complained of upper abdominal pain. He was examined by a doctor that morning and showed both hepatomegaly and splenomegaly, the liver being firm and

* The authors acknowledge the cooperation of Dr. I. H. Mathieson, deputizing for the District Surgeon, Middelburg, C.P., for obtaining this specimen.

tender. The convalescence of this individual was protracted in that he continued feeling discomfort in his abdomen although the liver and spleen were no longer palpable.

The blood specimen from P. was stored on water ice for 2 days. After this the serum was separated and inoculated intracerebrally into 2 litters of newborn and a group of 6 adult mice. There were no deaths or signs of illness in the inoculated mice during the 21-day period they were observed. A 1-in-10 dilution of the serum in bovine plasma-albumin was re-inoculated intracerebrally into a litter of mice with no evidence of pathogenic effects.

This acute-phase specimen of serum and one obtained from P. 42 days later were tested against H 112 virus in an intracerebral adult-mouse neutralization test. Each serum was heat-inactivated for 30 minutes at 60°C before the testing. The convalescent specimen neutralized 3·3 logs of H 112 virus in the test controlled by the acute-phase serum. Such an increase in neutralizing antibodies was taken as firm evidence that the aetiologic agent of illness in the second case (P.) was the same virus that was isolated from de M.

DISCUSSION

In the two cases of Wesselsbron-virus infection cited in this paper and those reported elsewhere,[1, 2] there have been no pathognomonic signs or symptoms elicited during the course of the illness. The total number of cases with the diagnosis confirmed by virus isolation or a significant immune response in convalescence has been small. The clinical spectrum may thus be broader than is indicated by these reports.

There are certain epidemiological factors that might assist the clinician in his attempt to determine the aetiology of such cases. Illness due to Wesselsbron-virus infection acquired under natural conditions would be expected to have a seasonal incidence. This period would be expected during the warmer months and especially if rainfall had been above normal. Both factors favour higher mosquito populations.

The mode of transmission might be direct contact with infected tissues or the bite of a vector mosquito. Wesselsbron virus has been isolated from several species of wild-caught mosquitoes collected in the Union of South Africa.[2, 5] In the laboratory two of these species have been shown to be capable of transmitting the virus by bite after a significant intrinsic incubation period (Muspratt *et al.*[3] and Kokernot *et al.*[6]).

There appears to be a rather widespread geographical distribution of Wesselsbron virus infection in man and domestic animals in southern Africa. The virus has been isolated in the Free State, in northern Natal and in the eastern part of the Cape Province. Serological surveys for the presence of neutralizing antibodies to this virus in the sera of indigenous human beings and domestic animals of northern Natal indicate a high incidence of immunity.[7] Weiss *et al.*[1] report the finding of antibodies in bovine sera collected at Knysna and also in the Rhodesias.

A third factor of epidemiological significance is the patient's occupation. This virus has been responsible for epizootics among the sheep population in South Africa. Therefore individuals engaged in sheep husbandry have a greater degree of exposure to infection with the agent, both in handling infected animals and in their exposure at work in the infected zone.

The aetiology of Wesselsbron virus infection in man can be determined with certainty only if the virus is isolated or if there is a significant rise in titre of antibody in a convalescent serum when compared with a specimen taken during the acute phase of the illness. To attempt virus isolation the blood specimen should be obtained in the early stage of illness and during the period of pyrexia, as the period of viraemia in arthropod-borne virus diseases is short. In the second case (P.) reported in this paper the blood was taken soon after the termination of pyrexia and virus isolation was unsuccessful. If the diagnosis is to be made by serological study it is necessary to have paired sera. The first should be taken as early as possible in the course of the illness and the second during the 2nd or 3rd week of convalescence. Both specimens should be refrigerated without actual freezing and handled in such manner as to prevent bacterial contamination and minimize haemolysis.

SUMMARY

1. Two clinical cases of Wesselsbron-virus infection in man are described.

2. The diagnosis was confirmed by virus isolation in one case and by significant increase in titre of neutralizing antibodies in both cases.

3. Epidemiological factors are discussed which might assist the practitioner in making a clinical diagnosis.

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STUDIES ON THE TRANSMISSION OF WESSELSBRON VIRUS BY *AEDES (OCHLEROTATUS) CABALLUS* (THEO.)*

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During the study of an epizootic in the eastern Cape Province, Wesselsbron virus was isolated from sheep (R. A. Alexander[1]). In the course of subsequent investigations by us on the farm Crowboroughvlei undertaken principally to identify the vector or vectors of the disease, further isolations were made from sheep, from 2 species of mosquitoes (Kokernot *et al.*[2]) and from man (Heymann *et al.*[3]). *Aedes* (*Ochlerotatus*) *caballus* (Theo.) was the prevalent mosquito and one of the two species from which we isolated Wesselsbron virus. The purpose of this report is to present results which demonstrate the ability of *Aedes caballus* to transmit that agent.

METHODS AND MATERIALS

With only the few exceptions mentioned below the methods and materials employed in these experiments were the same as those previously described (Kokernot *et al.*[4]).

A lamb (AN 2348) obtained on 9 April 1957 from Grootfontein Agricultural College, Middelburg, Cape Province, was exposed to 40 *Aedes caballus* females caught in the epizootic area the afternoon of the same day. The head and neck of the lamb were placed through the sleeve of a gauze cage measuring 14 × 14 × 14 inches. The legs of the animal were secured to assure restraint for a period of approximately 1 hour between 18.00 and 19.00 hours. During this interval 6 of the mosquitoes engorged and were pooled and designated AR 731. The remaining 34 mosquitoes showed no visible evidence of having engorged and were pooled and designated AR 732. Both lots were stored on dry ice overnight and the following day processed and inoculated into litters of newborn and adult mice in Johannesburg.

Immediately after exposure the lamb and mosquitoes were transported by car in an overnight journey to Johannesburg.

Aedes caballus that had been reared in the laboratory from larvae were allowed to engorge on lamb AN 2348 on 2 occasions. The first time was 46 hours after the initial exposure to AR 731, when the lamb's temperature was 104·4°F, and 14 of the laboratory-reared *Aedes caballus* (lot 35) engorged through the gauze covers of glass vials in which they were confined. On the third day, when its temperature was 104°F or over, the lamb was placed inside a plastic-screened cage measuring 30 × 24 × 21 inches, and in a 2-hour period from 20.00 to 22.00 hours 6 laboratory-reared *Aedes caballus* (lot 36) engorged.

Other *Aedes caballus* caught biting man in the epizootic area on the afternoon of 9 April were collected in glass tubes lined with strips of saturated absorbent paper and transported alive to Johannesburg. The next day 134 of these were observed to probe or engorge on 31 newborn mice held firmly over the orifices of the tubes. Each mouse was thus bitten by more than one mosquito. After exposure, the infant mice were returned to their respective mothers. The mosquitoes were then pooled in lots of 50, 50 and 34 and designated AR 749, AR 750 and AR 751 respectively. Each lot was killed by being placed at −20°C and then processed in the usual manner.

* The studies and observations on which this paper is based were financed jointly by the South African Institute for Medical Research, the Poliomyelitis Research Foundation, the South African Council for Scientific and Industrial Research, the Union Health Department, and the Rockefeller Foundation, and were conducted with the collaboration of the Veterinary Division of the Department of Agriculture.

RESULTS

Wesselsbron virus was isolated from the 6 *Aedes caballus* (AR 731) that engorged on lamb AN 2348 on 9 April 1957, and from the 34 (AR 732) showing no evidence of engorgement.

On the 2nd, 3rd and 4th days after exposure to 40 *Aedes caballus* mosquitoes (AR 731 and AR 732), lamb AN 2348 had a temperature in excess of 104°F. The 4th day it was anorexic, dyspnoeic and weak. Death occurred on the 5th day. Lots 35 and 36 *Aedes caballus* were allowed to engorge when the temperature was in excess of 104°F and during a period when Wesselsbron virus was circulating, as confirmed by subsequent specificity test. These details are shown in Table I.

TABLE I. COURSE OF WESSELSBRON INFECTION IN LAMB AN 2348 WHICH WAS SOURCE OF INFECTIVE FEED FOR LABORATORY-REARED *Aedes caballus*, LOTS 35 AND 36

Date	*Hour*	*Temperature*	*Remarks*
9.4.57	18.00	Not recorded	Exposed 1 hour to 40 wild-caught *Aedes caballus* collected 9.4.57 biting man.
11.4.57	08.30	100·2°F	
	12.00	101·6	
	15.50	104·4	14 *Aedes caballus*, lot 35, engorged; lamb then bled and Wesselsbron viraemia confirmed.
	22.15	103·6	
12.4.57	08.25	102·8	
	12.00	103·8	
	16.00	105·3	Confirmed Wesselsbron viraemia.
	20.00	104·4	During this 2-hour interval 6 *A. caballus* lot 36, engorged.
	22.00	104·0	
13.4.57	08.50	102·7	
	11.50	102·8	
	16.15	104·3	Anorexic, dyspnoeic and weak.
14.4.57	08.15	101·3	Critically ill.
	12.00		Died—Wesselsbron virus isolated from liver, kidney and spleen.

On the 19th post-feeding day one of the 14 *Aedes caballus* in lot 35 died. A suspension was made of this mosquito and inoculated into infant mice. An identification test confirmed the presence of Wesselsbron virus in this mosquito (Table III, experiment 2). After the lapse of 21 and 22 days, selected

TABLE II. FATE OF NEWBORN MICE BITTEN BY *Aedes caballus* WHICH HAD PREVIOUSLY ENGORGED ON A LAMB (AN 2348) CIRCULATING WESSELSBRON VIRUS

Mosquito lot no.	*Interval between feeding on lamb and mice*	*Number of mosquitoes biting mice*	*Apparent transmission rate*	*Fate of mice*
35	21 days	6	1/7	1 moribund 9th day—confirmed transmission (Table III, experiment 3). 6 remained well.
36	22 days	3	7/7	1 sick 7th day. 6 sick 8th day Confirmed transmission by means of pooled brains of 2 mice.

mosquitoes of lots 35 and 36 respectively were allowed to bite newborn mice. The results are summarized in Table II. Although 6 mosquitoes of lot 35 were utilized in biting newborn mice many times, only one mouse developed illness during the 21-day observation period. The illness in this mouse was confirmed as being due to Wesselsbron virus (Table III, experiment 3). The 3 mosquitoes of lot 36 bit another group of 7 newborn mice with 100% apparent transmission rate. Two of these mice, sick on the 8th post-biting day, were sacrificed and their brains pooled and then set up in a neutralization test to confirm Wesselsbron virus transmission. Over 4 logs of the agent in the pooled mouse brains was neutralized by a post-inoculation Wesselsbron guinea-pig serum when compared to the pre-inoculation serum.

Thus, laboratory-reared *Aedes caballus* from 2 lots differentiated by the time and method of exposure for engorgement on a lamb circulating Wesselsbron virus were able to transmit the virus to infant mice. The details confirming Wesselsbron virus as the agent involved in a successful lamb—*Aedes caballus*—mouse transmission are shown in Table III.

In addition to the transmission of Wesselsbron virus by the technique described above with laboratory-reared *Aedes caballus*, it was also transmitted by wild-caught mosquitoes (AR 749, AR 750 and AR 751). However, only one of the 31 mice bitten 2 to 4 times by a group of 134 such mosquitoes sickened. In a neutralization test the aetiology of the illness as having been due to Wesselsbron-virus infection was confirmed.

The three lots into which the 134 wild-caught *Aedes caballus* were divided for processing after having bitten mice (lots nos. 749, 750 and 751) yielded 4 strains of virus. From AR 749 a mixture of Middelburg (Kokernot *et al.*[4]) and Wesselsbron viruses was isolated. Wesselsbron virus was isolated from AR 750 and Middelburg virus from AR 751.

DISCUSSION

Wesselsbron virus was isolated from the wild-caught *Aedes caballus* (AR 731) that fed on lamb AN 2348 and this animal was subsequently proved to be infected with that virus. Nevertheless, this cannot be regarded with certainty as an experimental transmission because the lamb was obtained from a flock of sheep in which morbidity and mortality due to Wesselsbron virus were occurring.* When obtained, the lamb appeared healthy, but it is possible that Wesselsbron virus infection, acquired from a different source than mosquitoes of lot AR 731 or AR 732, was in an incubation phase. Regardless of the source of the lamb's infection, the Wesselsbron virus which was present in its blood served as the infecting feed for 2 lots of laboratory-reared *Aedes caballus* (lots 35 and 36).

A successful transmission by means of mosquitoes collected from nature within such a short period as 24 hours might include specimens capable only of mechanical transmission. However, the results presented include successful transmission by laboratory-reared *Aedes caballus* after a 21- and 22-days post-infective feeding period. There is a significant difference between the transmission rate to mice by bite of mosquitoes in lots 35 and 36. Lot 35 mosquitoes were exposed to AN 2348 a day earlier in the course of the illness, and it is probable that at that time the viraemia level was at a low infective threshold. The two lots of mosquitoes were exposed to the lamb by different methods. Lot 35 mosquitoes were maintained individually in glass vials. They were allowed to probe and feed upon the lamb through the gauze-covered orifice. Lot 36 mosquitoes became engorged with blood by feeding upon the lamb restrained in the cage with the mosquitoes.

The significance of *Aedes caballus* as a vector of Rift Valley fever virus (Gear *et al.*[5]) and Middelburg virus (Kokernot *et al.*[4]) has previously been reported. It is apparent from the current results that it is also a vector of Wesselsbron virus. The bionomics of this little-studied species merits intensive investigation with the hope that some practical method of control will be revealed.

Aedes caballus is the second member belonging to this

* The assistance of Dr. C. W. A. Belonje, Senior State Veterinarian, in the collection of field specimens from sheep is gratefully acknowledged.

TABLE III. RESULTS OF 3 NEUTRALIZATION EXPERIMENTS THAT CONFIRM WESSELSBRON VIRUS AS BEING THE AGENT IN A SUCCESSFUL LAMB-*Aedes Caballus* (LOT 35)-INFANT MOUSE TRANSMISSION

Experiment No.	*Source of infected infant mouse brain*	*Serum pre- or post-Wesselsbron virus**	*Dilution of infected brain suspension added to equal volume of serum*	*Results (Fate of mice)* Died	Lived	*Titre*
1. Identity of agent circulating in blood of lamb AN 2348 which was the source of infective feed for lot 35 *Aedes caballus*	Mice inoculated with serum taken from AN 2348 immediately after feeding 14 *Aedes caballus*, lot 35	Pre-	10^{-3}	3	0	3·86
			10^{-4}	5	7	
			10^{-5}	0	12	
			10^{-6}	0	12	
		Post-	10^{-1}	0	6	0·5†
			10^{-2}	0	6	
2. Identity of agent present in 1 *Aedes caballus* on the 19th post-feeding day	Mouse inoculated with the mosquito suspension	Pre-	10^{-3}	12	0	4·6†
			10^{-4}	12	0	
			10^{-5}	1	5	
			10^{-6}‡			
			10^{-7}	0	6	
		Post-	10^{-1}	0	12	0·5†
			10^{-2}	0	10	
3. Identity of agent causing illness in 1 of 7 infant mice bitten by *Aedes caballus* on their 21st post-feeding day	Mouse moribund on the 9th post-mosquito biting day	Pre-	10^{-3}	6	0	5·17
			10^{-4}	12	0	
			10^{-5}	6	6	
			10^{-6}	2	4	
			10^{-7}	0	6	
		Post-	10^{-1}	0	12	0·5†
			10^{-2}	0	10	

* Sera in experiments 1 and 2 were from guinea-pigs and serum in experimen 3 was from a sheep.
† Approximate titre since no clear end-point could be calculated.
‡ Unsatisfactory litter

genus shown in this laboratory to be capable of transmitting Wesselsbron virus. Muspratt *et al.*[6] demonstrated the ability of *Aedes* (*Banksinella*) *circumluteolus* to transmit the virus. Furthermore, the virus has been isolated in nature from the latter species (Smithburn *et al.*[7]).

SUMMARY

1. Laboratory-reared *Aedes caballus* were allowed to engorge on a lamb circulating Wesselsbron virus.

2. On the 21st and 22nd post-engorgement days these mosquitoes transmitted the virus by bite to new-born mice.

3. 134 wild-caught *Aedes caballus* were allowed to bite 31 new-born mice. From this a successful transmission resulted and Wesselsbron virus was confirmed in the single mouse showing illness.

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PARALYTIC MUSSEL POISONING IN NATAL

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Paralytic mussel poisoning has been reported from many parts of the world.[1] Most of the literature refers to the Pacific Coast of North America, where extensive investigations have been conducted.[2] American workers[3] have identified the neurotoxin as an alkaloid occurring in a dinoflagellate plankton, *Gonyaulax catenella*, on which the mussels feed. Sapeika[4] first described its occurrence in South Africa and also isolated the alkaloid. A recent leading article[5] noted that cases had not been reported for some time. We report here the poisoning of a Zulu family by mussels eaten on the coast of Natal, 35 miles north of Durban.

DESCRIPTION OF CASES

On the morning of 9 December 1957 a young Zulu mother of Tongaat collected mussels from rocks exposed by the low tide. She killed the mussels by heating them without added water, extracted them from their shells and washed them in rain water to remove sand. She then cooked them in a well-known vegetable oil, adding onion and curry powder for flavouring. In the evening, the mussels were eaten with dry maize porridge by herself, her husband, their 2 sons aged 8 and 3 years, and their daughter aged 5 years.

Onset of Symptoms

Less than 1 hour after the meal, the mother felt a peculiar lightness of her body, with a tingling around her mouth and in her fingers and toes. Unperturbed by these symptoms she went to bed with the rest of the family. About 6 hours after the meal she was awakened by her daughter calling out that she wanted to pass urine but could not move. The mother got up to find herself weak. In lighting the paraffin lamp her hand wavered. She said she had 'to pull herself together' before she could apply the match to the wick.

She managed to drag her daughter from the bed and had to support her whilst she passed urine. She then noticed that the child's breathing was laboured and that her attempts to speak resulted only in feeble inarticulate noises.

She awakened her husband and sent him to summon help. He did not feel ill but noticed that his body felt light and as he walked he had to concentrate hard to keep on the road. The mother also awakened both her sons and found that the eldest boy was hardly able to walk and his brother could neither sit up nor stand. She returned to her daughter, only to find her dead in bed.

Before leaving for hospital they noticed that a cat which had eaten the remnants of their meal was dragging itself about on its belly, owing to apparent loss of power in its lower front legs and hind legs.

Clinical Observations

Approximately 16 hours after the meal, the family was admitted to hospital. Both husband and wife complained of a feeling of lightness and tingling in their extremities and around their mouths. Clinical examination was, however, negative and both were symptom-free within 24 hours of eating the mussels.

The elder boy had a temperature of 99·8°F and weakness of the erector spinae muscles only. He had recovered completely 42 hours after eating the meal.

The younger boy's temperature was 100°F. He had a flaccid paresis of all four limbs and the erector spinae muscles were paralysed. The action of the intercostal muscles appeared weak. The deep reflexes were absent. He reacted normally to pinprick. The cerebrospinal fluid was normal. No treatment was given but a respirator was held in readiness. Within 24 hours he had improved. His temperature was normal and he could roll over in his cot and move all four limbs against gravity. However, it was not until the 5th day that he was able to sit up unaided. Weakness of the quadriceps femora was a marked feature, and to stand he had to climb up his legs like a child with pseudo hypertrophic myopathy. It was 14 days before he had recovered completely.

We saw the cat 4 days after it had eaten the mussels and it was then moving normally.

Investigation of the Mussels

The site of the poisoning was visited 4 days afterwards. There was no evidence of the red discolouration of the sea caused by the plankton floating on the surface which has been described by other workers,[1, 2, 4] nor had this ever been noticed by the local inhabitants. Mussels were collected from the same rocks and submitted for examination to Mr. W. Pople, of the Zoology Department, University of Natal, to whom we are indebted for this report:

'The mussels were the South African Black Mussel (*Mytilus meridionalis*). Attempts to recover a dinoflagellate from the crop contents and faeces were unsuccessful, but minute dinoflagellates were discovered in the rejection currents from the gills of all the mussels. These flagellates measured

3–4μ, which is the same size as *Gonyaulax catenella*,[6] but with the equipment available it was impossible to identify them exactly. Permanent slides were therefore made.

'After the mussels had been in filtered sea water for 6 days, 6 were chosen for testing. By the method described by Dack[7] two extracts were made from these mussels. One was made from the digestive glands and was brown in colour, the other, from the gills, was cream coloured. These extracts were tested by injecting them into the dorsal lymph spaces of 20-g. toads (*Bufo regularis*).

'With the digestive gland extract it was found that 0·25 ml. caused paralysis of leg reflexes within 11 minutes and respiratory paralysis within 15 minutes. No effect was produced with 0·1 ml., while 0·5 ml. caused complete paralysis and death within 7 minutes. An injection of 0·5 ml. of the control gill extract produced no effect whatever.

'Interpreting these results in accordance with Sapeika's statements[8] that 10 times as much toxin is required to kill a frog as a mouse, and that 1 mouse unit is the quantity of toxin in 1 ml. of neutral solution required to kill a 20-g. mouse within 15 minutes, we can conclude that this extract has a toxicity well above 4,000 m.u. per 100 g. of mussel, which is considered significant by the American workers.[2]'

DISCUSSION

It is 10 years since an outbreak of paralytic mussel poisoning occurred at Blaauwberg Strand, near Cape Town[4, 9]. From the enquiries we have made, it appears not to have been experienced before in Natal, although mussels have always been a popular food.

Three other types of mussel poisoning have been described, namely, various allergic reactions, gastro-enteritis from eating mussels that are not fresh, and occasionally typhoid fever from mussels infected by sewage. However, death from these illnesses is uncommon. It is a significant fact that both now and in 1948, when paralytic poisoning has been encountered, deaths have occurred. The alkaloid which causes paralytic mussel poisoning accumulates in the digestive glands of the mussels, which are themselves unaffected by its presence. It is water-soluble and heat-stable in acid or neutral solution, but is rendered inert when boiled in an alkaline solution. Sapeika[8] has shown that in addition to a central and neurotoxic action there is a depressant action on the myocardium and its conducting tissues.

The symptoms and signs in our cases are similar to those described previously. The cause of death is respiratory paralysis, and this probably occurred in the girl. The pathology of this condition has not been adequately studied, and it is unfortunate that no autopsy was possible in our fatal case.

There is no known antidote to paralytic poisoning and accordingly the only measures which can be taken are preventive. There is no way of telling whether mussels contain the toxin, as it occurs in healthy fresh specimens. The only warning is the redness of the sea when the dinoflagellates abound. It is recommended therefore that all mussels for human consumption should be boiled in water containing bicarbonate of soda,[4] in order to destroy the toxin which may be present. It should also be noted that since plankton floats on the surface, any other filter-feeding invertebrates such as sea-lice and rock oysters (*Ostrea cuculata*) which, like mussels, live between the tide levels, can accumulate this toxin.[10]

Symptoms occur soon after eating affected mussels and therefore an immediate stomach wash-out in suspected cases is imperative. Once paralysis is present the danger of respiratory failure has to be anticipated and the necessary apparatus for artificial respiration held ready. Pressor drugs such as ephedrine have been shown, experimentally, to combat myocardial depression.[8]

SUMMARY

An account is given of paralytic mussel poisoning in a Natal Zulu family, with the death of a child.

A dinoflagellate was recovered from the mussels, and from their digestive glands a potent neurotoxin was extracted.

The potential danger of eating fresh healthy mussels is stressed. This danger may be obviated by boiling them in a solution of sodium bicarbonate.

We are grateful to the Medical Superintendent, Dr. S. Disler, for permission to publish this paper, Dr. H. L. Wallace for his critical advice, Dr. S. Goldberg for his assistance and Dr. J. Kelman Drummond, Dr. N. R. Pooler, and Dr. N. MacE. Lamont for access to cases under their care.

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THE EFFECT OF VARIOUS DIETARY FACTORS ON THE SERUM-CHOLESTEROL LEVEL AND ON THE FAECAL FAT CONTENT*

H. GORDON, B.Sc., M.B., CH.B., J. WILKENS, B.Sc. (HONS.) AND J. F. BROCK, M.D., F.R.C.P.

From the C.S.I.R./U.C.T. Clinical Nutrition Research Unit, Department of Medicine, University of Cape Town; and the Groote Schuur Hospital

A. The effect of some non-lipid dietary factors on the serum-cholesterol level

Carbohydrate. Two subjects were studied. Initially a 2,500-calorie, almost fat-free diet was administered; then supplements of sucrose were added to increase the caloric intake to 3,500 daily for 30 days and to 4,000 daily for a further 25 days. Moderate increases in the serum-cholesterol level occurred only during periods of rapid gain in weight. When saturated fats were substituted for sucrose at the same or at lower caloric levels, there were prompt and substantial increases in the serum-cholesterol level. These observations are consistent with the theory that the serum-cholesterol level rises when there is an increase in the fat transport load, e.g. at the time of rapid gain in weight, when there is increased lipogenesis from carbohydrate.

* Abstract of paper presented at a meeting of Research Forum, University of Cape Town, held at Groote Schuur Hospital, Cape Town, on 7 May 1958.

2. *Protein.* Three subjects were studied. Iso-caloric diets were maintained throughout the experimental period and the intake of protein was varied by feeding with different quantities of maize meal or skimmed-milk protein. Provided that the fat consumption was constant, changing the daily protein intake from 34 g. to 68 g. did not alter the serum-cholesterol level. When milk fat was isocalorically substituted for carbohydrate at these protein levels, the serum-cholesterol level rose promptly and substantially.

3. *Cellulose.* The effects of diets containing different quantities of crude fibre were studied in short-term experiments on 3 subjects. The diets were constant throughout the experiments with regard to calories, protein and quantity and quality of fat. In 2 subjects, changing the daily consumption of crude fibre from 6·1 g. to 14·5 g. by increasing the intake of maize meal did not affect the faecal fat content or the serum-cholesterol level. In a 3rd subject, the dietary fibre was increased to 24·5 g. daily by the addition of coarsely milled, hexane-extracted sunflower-seed hulls; this highly artificial fibre load produced a slight increase in the faecal fat content and a slight fall in the serum-cholesterol level.

B. Studies to determine the origin of the faecal fat

In 11 subjects studied on basal diets very low in fat (6·5 g. to 8·6 g. daily) for an average of 13 days each, the mean daily faecal fat content ranged from 0·8 g. to 5·4 g. with a mean of 2·25 g. (S.D. 1·12). This fat is presumably derived from a combination of synthesis by intestinal bacteria, desquamation of intestinal mucosal cells, and excretion *via* the biliary system or through the intestinal wall. There was no correlation between the faecal fat content and the dietary fibre intake, the latter varying among the subjects from 1·8 g. to 6·8 g. daily. In the 2 subjects described above, increasing the dietary calories from 2,500 to 4,000 did not materially influence the faecal fat content. In 15 subjects, the addition of saturated or unsaturated fat to the basal diet always produced an increase in the faecal fat content; in general, the greater the dietary load of fat, the greater was the faecal fat content. To determine whether this increase in faecal fat content was due to unabsorbed dietary fat or to increased excretion of body fat, a series of experiments was conducted in 5 subjects with intravenously administered emulsions of cottonseed oil (Lipomul I.V.—Upjohn). When the intravenous fat was administered instead of dietary fat, the faecal fat content fell towards basal levels; when it was administered in addition to the dietary fat, there was no further increase in the faecal fat content. These observations are consistent with the hypothesis that with ordinary diets the faecal fat is derived from unabsorbed dietary fat and not from active excretion of body fat. It was noted that in all these subjects the administration of intravenous cottonseed oil produced a decrease in the serum-cholesterol level, but when the emulsifying agents alone (soybean phosphatide and pluronic F68) were administered, without the cottonseed oil, the serum-cholesterol level always rose. This apparently is an example in man of the phenomenon described in experimental animals of 'cholesterol trapping' in the blood stream by emulsifying agents.

C. *Studies to determine the cholesterol-decreasing factor in certain unsaturated fats*

The following preliminary observations were reported:

1. The unsaponifiable fractions of pilchard oil and sunflower-seed oil failed to reproduce the cholesterol-decreasing effect of the fatty-acid fractions of the whole oil when eaten by 3 subjects.

2. In a series of individual experiments in the metabolism ward and in a group experiment on 30 men in an institution, natural and hydrogenated oils were compared. The hydrogenating of maasbanker oil so as to reduce its iodine value from 170 to 70, peanut oil from 89 to 55, and sunflower seed oil from 135 to 74, abolished cholesterol-decreasing action of these fats. There did not, however, appear to be a linear relationship between the iodine value of the fat and its effect on the serum-cholesterol level.

3. To test further the effect of altering the iodine value, a series of experiments was devised in which hens' eggs were 'unsaturated' so as to increase the iodine value from 72 to 100. This was done by feeding a flock of Black Australorp hens with a diet rich in sunflower seeds. Ten yolks daily of 'unsaturated' eggs and 'control' eggs were eaten by 2 subjects for 10 days; no difference was noted in their cholesterogenic effect. As has previously been shown, this effect of the 'unsaturated' eggs was not due to their cholesterol content and it was again shown that this effect could be abolished by frying the eggs in sunflower-seed oil.

Our results thus far do not support the theory that the action of all dietary fats on the serum-cholesterol level is determined simply by their degree of saturation. Further investigations are in progress in order to characterize more precisely the active factor or factors.

IN MEMORIAM

DR. JACK GREENSTEIN, M.D. (HONS.), B.CH., L.R.C.P. & S. (EDIN.), L.R.F.P.S. (GLAS.)

Dr. J. S. Zidel, Johannesburg, writes: Dr. Jack Greenstein died in Johannesburg on Easter Monday, 7 April 1958. He was only 57 years old.

Jack Greenstein commenced his medical education at the University of the Witwatersrand, and thereafter continued at Edinburgh University, where he qualified in 1925. After qualifying he set up practice in Lichtenberg, in the Western Transvaal, where he built up an extensive practice, with a reputation far beyond the borders of the district, and contributed a great deal to cultural life.

Dr. Jack Greenstein

In 1938, he went to England and studied at the British Postgraduate School of Medicine in Hammersmith, where he undertook extensive investigations into the incidence and causation of thrombosis and pulmonary embolism. He returned to Johannesburg after the outbreak of war, and accepted an appointment as District Surgeon and Lecturer in Forensic Medicine at the University of the Witwatersrand. This enabled him, with the permission and encouragement of the then Head of the Department, Professor McIntosh, to continue his investigations into circulatory disease. He did a great deal of valuable work and published several papers on his researches, among them 'Thrombus Formation in the First Part of the Aorta and Coronary Embolism—its Medico-Legal Aspect', and 'Comparative Causes of Sudden Death in the European and Bantu'.

In 1945 he returned to Britain for further study, and was awarded his M.D. with Honours at Edinburgh University for a thesis based on his earlier studies, 'Deep Vein Thrombosis and its Relation to Pulmonary Embolism'. After Britain he spent a year in the United States, pursuing his studies in cardio-vascular disease at the Mayo Clinic, the Mount Sinai Hospital, and other well-known institutions, where he developed a special interest in phonocardiography.

On his return to South Africa he joined the staff of the newly-established cardiac and peripheral vascular clinics at the Johannesburg Hospital, where he applied his knowledge of phonocardiography and recorded a survey of 1,500 cases. In 1946 he commenced practice as a consulting physician in Johannesburg.

Dr. Greenstein was Senior Physician to the Discoverers' Hospital on the West Rand, and on the staff of the Cardiac and Peripheral Vascular Clinics of the Johannesburg General Hospital.

Jack Greenstein was an indefatigable worker, a shrewd observer, and a clear thinker. He was a man of very wide interests, particularly in literature and music. He was a warm, genial and lovable personality, a desirable friend, and a wise, painstaking and thorough physician. His opinions were based on deep wisdom and understanding in addition to wide reading and the critical evaluation of new sources of knowledge. He practised in the spirit of the saying, 'Man is born to live not for himself but for others'.

I, personally, in the role of patient, have much cause to be

eternally grateful to him for the devoted manner in which he applied himself unsparingly on behalf of his patients. I shall never forget his distress when the prognosis looked rather gloomy; how he explored every avenue of treatment, inviting every colleague who might be of assistance in my case, and the magnificant manner in which everyone responded to his call.

He will be missed by many who will retain the memory of his sincerity and modesty. An incident at the centenary of the American Medical Association, which he attended, will illustrate this. Jack Greenstein was listening to a lecture in the cardio-vascular section. He had arrived after the lecture had begun. The lecturer was quoting statistics, and Greenstein was struck by the remarkable fact that the lecturer's figures were identical with his own. It was only some time later that he discovered that the figures quoted were in fact Greenstein's.

Our heartfelt sympathy goes out to his widow, Bessie, and to his children, Dr. Adrian Greenstein, Mrs. Marna Shapiro (wife of Dr. Sydney Shapiro), Felicity and Robert.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Sylvia C. Gavron, of the University of Cape Town Child Guidance Centre, has returned from a 5 months' leave of absence in England, Europe and Israel. Whilst abroad she attended child psychiatric hospitals and clinics, including Maudsley and Great Ormond Street Hospitals.

* * *

South African Society of Medical Women. The annual general meeting of the Cape Town Sub-group of this Society will be held in the Gynaecology Lecture Theatre in the Pathology Block of the Medical School, Observatory, Cape, on Monday 26 May at 5.15 p.m. Medical women wishing to join the Society are welcome to attend.

* * *

National General Practitioners Group, Cape Western Sub-Group. At the Annual General Meeting held on 30 April the following appointments were made: *Chairman* Dr. George A. Paterson, *Vice-Chairman* Dr. F. E. Hofmeyr, *Secretary-Treasurer* Dr. Norman Levy, *Assistant Secretary* Dr. Sid Kiel, *Committee*—Drs. C. H. Bösenberg, V. Fox, M. Geffen, A. Gordon, S. Schur, C. Shapiro, L. Sternberg, I. Wolfsohn.

Uniale Groep Algemene Praktisyns: Onderafdeling Wes-Kaapland. Op die Algemene Jaarvergadering gehou op 30 April was die volgende gekies: *Voorsitter* dr. George A. Paterson, *Ondervoorsitter* dr. F. E. Hofmeyr, *Sekretaris-Penningmeester* dr. Norman Levy, *Assistent-Sekretaris* dr. Sid Kiel, *Komitee*—drs. C. H. Bösenberg, V. Fox, M. Geffen, A. Gordon, S. Schur, C. Shapiro, L. Sternberg, I. Wolfsohn.

Research Forum, University of Cape Town. The next two meetings of Research Forum will be held on Wednesday 4 June and Tuesday 17 June in the large lecture theatre on A Floor, Groote Schuur Hospital, Cape Town. They will both be devoted to a report on a large co-operative study on diet, cholesterol, fibrinolysis and blood coagulation. This has been studied in Bantu males and in White males with and without overt evidence of coronary artery disease. In addition to the speakers, Miss A. Moodie and Drs. B. Kaplan, H. Nossel and V. Schrire cooperated in this work. The speakers on Wednesday 4 June will be Dr. H. Gordon and Dr. R. Lackner; subject, 'Diet, Cholesterol and Fibrinolysis'. On Tuesday 17 June Dr. C. Merskey will be the speaker; subject, 'Diet, Cholesterol and Coagulation.'

* * *

The Czechoslovak Society of Physiotherapy is holding an *International Congress of Balneology* in Marienbad on 8-13 September 1958. The main subjects will be (1) conservative treatment and balneolotherapy in urology, and (2) thermoregulation in physiatrics and balneology. One day, also, will be devoted to the subject of choledocholithiasis, with an excursion to Carlsbad. Papers will be read by well-known scientists from Bulgaria, France, Germany, Hungary, Poland, the Soviet Union, Sweden and Czechoslovakia. The official languages of the Congress are English, French, Russian, German, Czech and Slovak. All papers will be read in those languages. There will be simultaneous interpretation to and from the official languages.

Excursions and cultural and social events will be arranged and a special programme provided for family members of participants. Application and inquiries should be addressed to the Secretary of the International Congress of Balneology, Reznická 5, Prague 2, Czechoslovakia.

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CORRESPONDENCE : BRIEWERUBRIEK

EMOLUMENTS OF MEDICAL POSTS IN THE PUBLIC SERVICE

To the Editor: Having recently read Dr. J. J. du P. le Roux's theoretical argument on salary structure, I do feel that he has not adequately explained his remark that 'the prospects of obtaining a basic salary of £1,740 per annum have increased considerably'. Would Dr. le Roux therefore kindly show how medical men in the lower grades reach this 'Utopian' salary scale. Does it depend on (1) seniority or (2) medical knowledge and experience or (3) ability to pass a language test?

A Starving Medical Officer

5 May 1958

EMOLUMENTS OF MEDICAL POSTS IN THE PUBLIC SERVICE

To the Editor: As another '£1,380 (fixed)' medical practitioner, employed by a local authority, I wholeheartedly endorse the remarks of your correspondent '*£1,380*'[1] in the *Journal* of 26 April 1958.

Whichever way we turn we find that we cannot pierce the '1380' barrier. No cognizance is taken of responsibility, experience, years of service and *bona fide* application to the job. It may well be that the Union Health Department will be prepared to consider and accede to part-refund on salaries higher than the £1,380 paid to Local-Authority Clinical Medical Officers in the light of the abovementioned factors, but the snag is that local authorities are not compelled to pay increased salaries.

I therefore wish to make the approach that where considered necessary in the interests of the medical profession, the local authority and the public health, the central Government should make it obligatory on local authorities to pay salaries in excess of £1,380.

Should other medical practitioners in local-authority service find themselves hamstrung by the ridiculous £1,380 barrier, may I appeal to them to express their opinion in the medical press in the hope that the authorities will take heed and realize that we are as vital to the public health as the medical officers of health under whom we serve?

Anti-1380—fixed

5 May 1958

1. '£1,380' (1958): S. Afr. Med. J., 32, 459.

DR. CHARLES BERMAN IN AMERICA

To the Editor: I am sending you the following information as I believe it may be of interest to your readers:

Dr. Charles Berman, M.R.C.P., of Maraisburg, Transvaal, and Visiting Associate, Sloan Kettering Institute for Cancer Research and Special Fellow in Surgery, Memorial Hospital for Cancer and Allied Diseases, New York City, delivered the thirteenth James Ewing Memorial Lecture at the New York Academy of Medicine, 1 May 1958. His subject was 'Primary Carcinoma of the Liver'. Dr. Berman discussed the aetiology, pathology and treatment of primary carcinoma of the liver and presented a stimulating discussion of the various theories pertaining to the origin and incidence of primary carcinoma of the liver in man and animals. His lecture was illustrated by many slides.

The James Ewing Memorial Lectures are given annually at the New York Academy of Medicine. They are designed to contribute to the teaching of cancer among general practitioners and lay people, and aim to bring to the general practitioners the concepts of newer research and techniques in the diagnosis and treatment of cancer.

Before Dr. Berman delivered his lecture, he and Mrs. Berman, and the Consul General, Mr. T. J. Endemann, were entertained at dinner and cocktails by Dr. Elise Strang L'Esperance, Chairman of the James Ewing Memorial Committee and Founder of the Strang Cancer Prevention Clinics.

Elise Strang L'Esperance, M.D.
Chairman, James Ewing Memorial Committee

525 Pelham Manor Road
Pelham Manor 65, N.Y.

2 May 1958

APPEAL FOR EMBRYOLOGICAL MATERIAL

To the Editor: I shall be grateful if I may be permitted to make an appeal to the readers of the *Journal*. It is essential for our embryological research programme to have available a large number of foetuses and embryos. In the past, local medical practitioners have been most cooperative in sending foetuses to me and now I wish to make a special request for young embryos; they are particularly required for a projected investigation of the development of the anal and rectal region.

We are even fully prepared to hunt for the embryos in uterine scrapings if these are placed in a 2–5% formalin or even a saline solution and sent to us. Specimens are easily wrapped in cotton wool soaked in one of these solutions and could be put in a small tin which could be sealed with scotch tape, e.g. an elastoplast tin, and sent either by surface or air mail.

I shall be extremely grateful for such cooperation and will cover postage expenses. It will also be appreciated if a covering note with the possible age of the specimen and the age and race of the mother can be sent.

It need not be stressed that this type of research is quite impossible without the kind favours of those who are able to supply us with the specimens.

Ronald Singer

Anatomy Department
University of Cape Town Medical School
Observatory, Cape

12 May 1958

COMMONWEALTH MEDICAL ADVISORY BUREAU

To the Editor: The Commonwealth Medical Advisory Bureau was established by the Council of the British Medical Association ten years ago to provide a welcome for doctors visiting the United Kingdom from other countries of the Commonwealth and also to give any help and advice that they might require either before coming to this country or after arrival.

The Bureau was officially opened in July 1948, and the use that has been made of it since has shown that it provides a most valuable service. More than 10,000 doctors have sought advice, most frequently on post-graduate education, during these ten years and a similar number of visiting doctors and their wives have attended social functions arranged by the Bureau on behalf of the Association.

It is not my intention, however, to give a full account of the activities of the Bureau but to make use of the occasion of the tenth anniversary to remind your readers of its existence. I should be grateful therefore if you could find space in your journal for this letter so that all doctors who may be considering coming to the United Kingdom whether for study or for pleasure, may know something about the Bureau and also may know that any enquiry or a visit from them will be most welcome.

R. A. Pallister
Medical Director

Commonwealth Medical Advisory Bureau,
British Medical Association House
Tavistock Square, London, W.C.1

8 May 1958